RADICULAR PAIN DUE TO RUPTURED CERVICAL DISK

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CERVICAL intervertebral disks which rupture into the spinal canal may produce no neurologic symptoms if the hernia is small (fig. 1B), may merely compress a single nerve root if the nodule is small and laterally placed (fig. 1C), may result in compression of the spinal cord and one or both adjacent nerve roots if the protrusion is large (fig. 1E), or may compress the cord alone if the hernia is discrete and near the midline (fig. 1D). The type of ruptured cervical disk which first received attention from neurosurgeons was, for obvious reasons, the one causing compression of the spinal cord and simulating spinal cord tumor. Reports of operations for this type of ruptured disk, although under the erroneous title of chondroma, began to appear in the literature about 1925.[1] Most important of these contributions was that of Stookey[2] in 1928 in which he described, under the title "extradural ventral chondromas," 6 cases of cord compression and 1 case of compression of a single nerve root (C VII). The first case of ruptured cervical disk to be reported under its correct title of herniation of the nucleus pulposus was by Peet and Echols[3] in 1934. In this case the cord was compressed by protrusion of the disk between the third and fourth vertebral bodies.

The reported cases of ruptured cervical disk giving cord compression are less than 75 in number.[4] However, this is certainly not a true picture of its incidence. Probably every neurosurgeon has a collection of unreported cases. I have records of 3 cases which were verified at operation. The most spectacular of these was that

of M. P., a 25 year old stenographer, who entered Touro Infirmary on Oct. 24, 1941. For the preceding 10 months she had complained of pain in the neck and both shoulders. Four days before admission she had had a violent attack of sneezing which greatly increased the pain and caused it to radiate down both arms, particularly the left. This was soon followed by partial paralysis of the left leg, loss of sensation in the right leg, and retention of urine. At operation on October 28, the spinal cord was found to be displaced posteriorly by protrusion of the seventh cervical disk. Nothing was done to the disk; the dura was left open for decompressive purposes. The patient eventually returned to her stenographic work and was found, on March 8, 1946, more than 5 years later, to be in good health even though she has considerable residual neurologic disability.

A second patient, J. P. W., was operated on at Touro Infirmary March 21, 1944, three and one-half months after a cervical injury which caused numbness of the entire trunk and weakness of the right leg. His condition improved somewhat but he did not become free of pain in the neck and arms. Flexion of the neck would cause a sensation like an electric shock to run through his body and often this would be accompanied by falling because of momentary weakness of the legs. Because there was no roentgenographic evidence of fracture-dislocation of the spine, a diagnosis of ruptured cervical disk was made and verified at the time of decompressive laminectomy. It proved to be the sixth cervical disk. Two years later, on March 8, 1946, this man was well and at work.

The third patient, E. N. S., had bilateral seventh root compression and mild cord compression due to protrusion of the sixth cervical disk as the result of an automobile accident. She found that flexion of the neck would cause a sensation of numbness to pass through the trunk and all extremities. The hands were said to be weak and she complained of pain in the neck and arms and of constant numbness in the thumb and index finger on each side. Because the symptoms progressed in spite of skeletal traction, decompressive laminectomy was performed at Touro Infirmary on June 2, 1944, two months after the injury. An interview on March 9, 1946, 21 months later, disclosed that she was nearly free of symptoms and doing all her housework, although she felt an occasional shock in the left arm when lifting.

It might be appropriate to mention here that some cases of transverse myelopathy supposedly due to fracture-dislocation of the neck are in reality cases of cord destruction by an extruded intervertebral disk as pointed out by Cramer and McGowan[5] and others.[6]
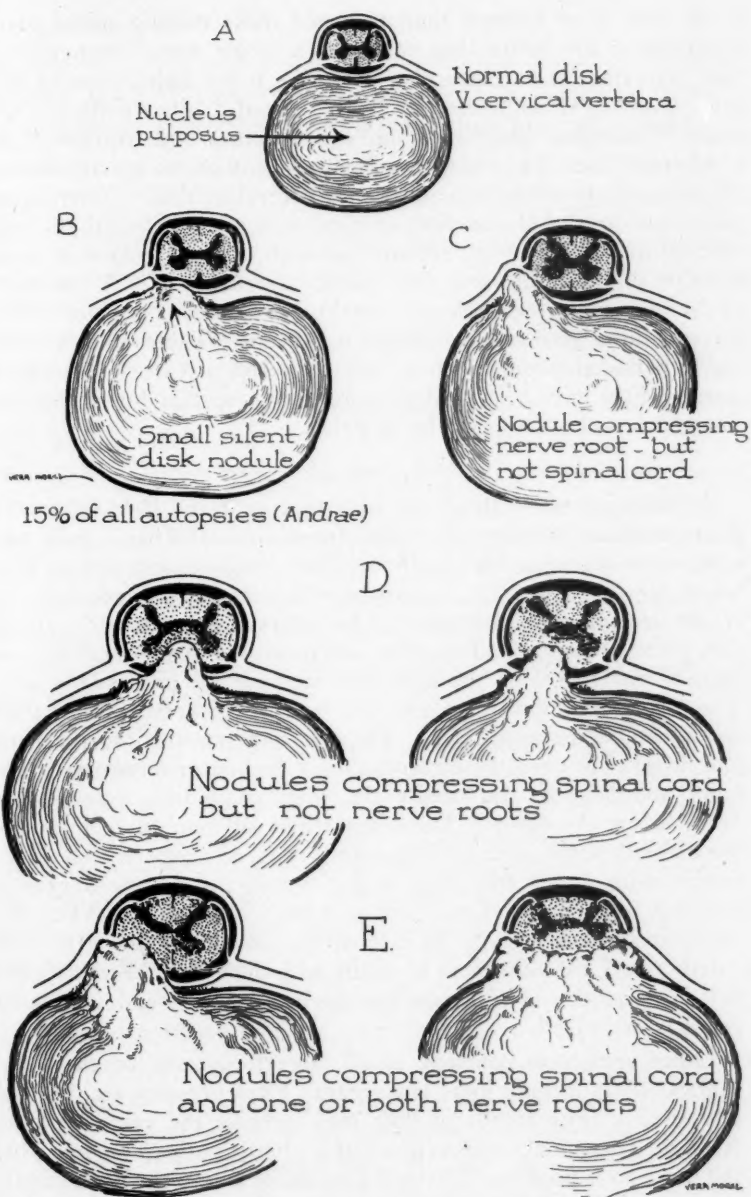
Fig. 1: Diagrammatic sketch showing a normal disk and the various types of disk ruptures. Three examples of type E and 3 examples of type C are reported in this paper.

Of even more interest than ruptured disks causing spinal cord compression are those that compress a single nerve root, giving pain, paresthesias and muscular weakness in the distribution of the root. Stookey's[2] single case of this type and 3 other scattered reports[7,8,9] attracted little attention until Semmes and Murphey[10] in 1943 established the syndrome of compression of the seventh nerve root by unilateral rupture of the sixth cervical disk. Other cases of compression of the seventh cervical root soon followed, as well as cases of rupture of the seventh and fifth cervical disks with compression of the eighth and sixth roots respectively.[11-15] A summary of the literature now discloses a total of 102 cases of rupture of the cervical disks giving the radicular syndrome (Table 1). According to personal communications, sizable series of cases of ruptured cervical disks have been studied in military hospitals during the last four years and are soon to be reported.

SYMPTOMS

As summarized by Bradford and Spurling in the 1945 edition of their excellent monograph, "The Intervertebral Disc," pain and stiffness of the neck are usually the first symptoms of rupture of a lower cervical disk. The symptoms referable to the neck may decrease or completely disappear to be followed by pain in the shoulder, pectoral region and scapula, and down the arm. Sneezing and certain movements of the neck may increase the pain in the arm. The points of maximum pain are the base of the neck, the tip of the shoulder and the upper arm. The pain may extend into the hand and fingers but paresthesias, including a feeling of numbness in the hand and fingers, are usually the chief complaints referable to areas below the elbow. These sensations of numbness in case of the disk below the sixth body (seventh root) are experienced chiefly in the index finger and often in the thumb and middle fingers as well.[10,11] The triceps muscle may be weak and its tendon reflex may be diminished or absent. In case of the disk below the fifth body (sixth root) the numbness is slight and chiefly in the thumb and first metacarpal. The biceps muscle is weakened and its tendon reflex is decreased.

Other symptoms common to all lateral ruptures of the lower cervical disks include local tenderness. Firm pressure on the back of the neck close to the midline may increase the radicular pain. Also, the foraminal compression test is almost pathognomonic when the reaction is positive. The test consists of tilting the head toward the painful side and applying pressure on the top of the head. It is positive if it reproduces or increases the radicular features of the syndrome.

· TABLE I

CASES OF CERVICAL ROOT COMPRESSION WITHOUT CORD COMPRESSION REPORTED IN THE LITERATURE

Date	Author	Cases	Opera-tions	Disk Below					
				7th Body	6th Body	5th Body	4th Body	3rd Body	2nd Body
1925	Adson[7]	1	1		1				
1928	Stookey[2]	1	1		1				
1937	Love & Camp[8]	1	1	1					
1941	Bradford & Spurling[9]	1	1			1			
1943	Semmes & Murphey[10]	4	3		4				
1944	Bucy & Chenault[13]	1	1		1				
1944	Michelson & Mixter[14]	8	8	1	3	4			
1944	Ulmer & Meredith[15]	2	2	2					
1944	Spurling & Scoville[11]	13	13		9	4			
1944	Epstein & Davidoff[16]	2	2	1	1				
1945	Elliott & Kremer[17]	8	0		8				
1945	Browder & Watson[4]	7	7	?	?	?	?	?	
1945	Young[18]	54	4	5	24	21	3		1
	Total	102	43	10	52	30	3	0	1

One of the symptoms common to irritation of any nerve root is muscle spasm, and this is a prominent feature in lateral rupture of

the cervical disk. However, the spasm is not always easy to detect and is best appreciated by abnormal postures of the head or by lateral films of the neck which invariably show loss of the normal cervical curvature. Since the scaleni muscles receive their innervation from the second to the seventh cervical nerves, it is not surprising that symptoms of scalene spasm, such as transient numbness of the ring and little fingers, may appear at times. However, it is not likely that the scalenus anticus syndrome is ever the direct result of a ruptured cervical disk.

Seventh Cervical Disk—Rupture of the lowest cervical disk (below the seventh body) has been reported only 10 times, but should in theory be the commonest of the disk lesions because it is the disk that joins the relatively inflexible thoracic spine with the freely movable cervical spine. It may be that some of the atypical cases of scalenus anticus syndrome with pain and paresthesia in the ulnar distribution are really cases of compression of the eighth cervical root by rupture of the seventh disk.

Sixth Cervical Disk—The sixth cervical disk is the one which ruptures most frequently. As previously mentioned, compression of the seventh cervical root by a nodule on this disk produces numbness and sometimes pain in the index finger and sometimes in the thumb and middle finger as well. Most of the pain is in the neck, thorax, scapula, shoulder, posterolateral surface of the upper arm, and dorsum of the forearm. The following is an example of such a case:

CASE 1: R. H. S., a business man, aged 51, entered Touro Infirmary on Nov. 21, 1942, because of severe pain in the left arm, shoulder, and side of the neck, which had begun immediately after a sudden movement of the head while hunting two weeks previously. However, he had been conscious of discomfort in the neck since a football injury sustained in college. For comfort he kept his head tilted away from the painful shoulder. The pain radiated down the lateral aspect of the arm and forearm to the hand and fingers. He also complained of tingling in the fingers but had difficulty identifying the exact fingers involved. A diagnosis of compression of the seventh root by a ruptured disk was made on the basis of hyperesthesia of the index finger and roentgenographic evidence of narrowing of the disk below the sixth cervical vertebra. Orthopedic measures, including traction followed by the wearing of a leather and metal collar, gave considerable relief. However, an attack of coughing two months later caused return of the symptoms. He found that he could increase the pain and paresthesia by certain movements of the neck. The results from conservative measures were discouraging this time and he eventually consented to operation on Feb. 23, 1943.

Under endotracheal anesthesia the sixth and seventh cervical laminae on the left were partially removed to expose the lateral aspect of the dural sac and the sheath of the seventh cervical root. The root was severely compressed

between a discrete disk nodule and the posterior margin of the intervertebral foramen. The root could not be retracted downward but it was possible to retract it in a cephalad direction sufficiently to incise the capsule of the nodule. The loose contents escaped spontaneously, as they frequently do in

Fig. 2: (Case 3.) A metal clip has been placed within the disk to demonstrate roentgenographically which disk was operated on. A second clip has been placed adjacent to the sixth root.

case of lumbar nodules. The bony ridges, reported in a high percentage of ruptured cervical disks, were either small or absent. Bleeding from the veins adjacent to the nodule proved to be a serious problem and 1,000 c.c. of blood was given intravenously. Postoperatively, the numbness and hyperesthesia in the index finger were greatly increased. The patient left the hospital on the ninth postoperative day. An interview on the third anniversary of the operation disclosed that he had no complaints.

An equally typical case of seventh root compression by a ruptured cervical disk is that of a patient who died during operation, the only tragedy of its kind with which I am familiar.

CASE 2: On Sept. 12, 1943, an unmarried, 44 year old office worker, felt something "pop" in her neck while stretching in bed. Pain immediately developed in the neck, shoulder and suprascapular region and down the arm to the tip of the index finger. A prominent symptom was numbness of the index finger and, to some extent, of the thumb and middle finger. Movements of the neck increased the pain. The diagnosis was obvious and supported by the roentgenographic findings, which included loss of the normal cervical curvature, slight narrowing of the disk below the fifth body and marked narrowing of the disk below the sixth body, together with osteophytic flanges which protruded into the spinal canal. Thirty-two days later the patient was operated on because orthopedic measures, including traction, had failed to give relief. About the time that the severely compressed seventh root was being unroofed by a chisel the patient died before the physician anesthetist realized that there was almost complete obstruction of the endotracheal tube. The pathologist, Dr. J. R. Schenken, completed the investigation in the autopsy room and found a disk nodule measuring 1 cm. in diameter. This nodule and 2 adjacent bony ridges encroached on the lumen of the intervertebral foramen and caused discoloration and pronounced flattening of the nerve root. Special stains showed demyelinization of some of the axones.

Fifth Cervical Disk—The disk below the fifth vertebra ruptures almost as frequently as the sixth. Such a lesion compresses the sixth root and produces sensory changes chiefly in the thumb and first metacarpal region. The biceps muscle may be weak and its reflex may be absent. The pain is felt along the radial aspect of the arm and forearm. A typical example of this lesion is as follows:

CASE 3: L. S., a physician, aged 30, registered at the Clinic on Aug. 6, 1942, because of recurring attacks of agonizing pain in the right upper arm and shoulder. Six years before, he had twisted his neck while swimming and suffered with stiffness of the neck for two weeks. Other attacks of stiff neck had occurred frequently thereafter. Such measures as head traction and plaster cast caused the pain to extend down the radial aspect of the forearm to the thumb. Pressure just to the right of the sixth spinous process, as well as certain neck movements, increased the pain. Although a diagnosis of ruptured disk with compression of the sixth cervical root was made at this time, the patient was unwilling to have an operation. Eventually he became addicted to morphine and was treated in a sanatorium. Nearly 3 years later another severe attack started while the patient was brushing his teeth. The pain was in the lateral aspect of the neck and in the shoulder and upper arm. There was no real pain below the elbow but there was tingling along the lateral aspect of the forearm and into the thumb. Some relief was obtained by holding the neck motionless and placing the hand behind the head.

The patient took morphine as required and reported for operation a few days later on March 23, 1945. Under endotracheal anesthesia the right fifth interspace was widened to expose the lateral aspect of the dural sac and the sheath of the sixth root. A discrete disk nodule was found anterior to the dural sac and the root. There were no adjacent bony ridges. The root was retracted upward sufficiently to permit incising the capsule of the nodule. A single large sequestrum was lifted out and the fissure leading to the center of the disk was probed. A silver clip was placed in this fissure for a roentgen-ray record (fig. 2). A clip was also left adjacent to the root.

On Feb. 20, 1946, 11 months later, the patient was found to be in excellent health and using the arm for all activities including fly-fishing. His only symptom is a vague discomfort between the shoulders toward the end of a long operation. This vanishes as soon as he stands erect. Roentgenograms
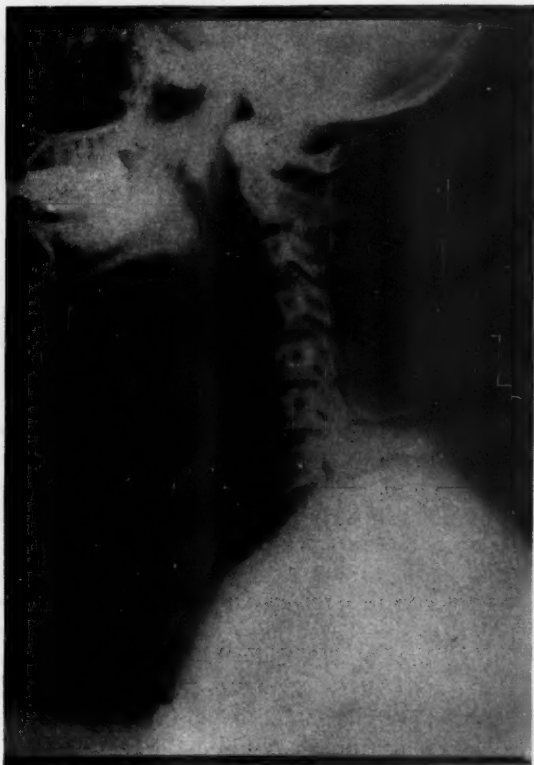

Fig. 3: (Case 3.) Lateral view of Case 3 (see fig. 2) showing metal clip inside the disk. The narrowing of this disk is scarcely perceptible.

show only questionable narrowing of the injured disk (fig. 3). Of even more interest is persistence of loss of the normal cervical curvature in spite of absence of symptoms.

Fourth Cervical Disk—Clear-cut examples of rupture of the disk below the fourth body have not been reported except for 3 cases listed in an article by Young.[18] Future case reports may clarify the details of the syndrome. From the anatomic point of view the pain and paresthesia should be in the neck, shoulder and arm.

Third Cervical Disk—Lateral rupture of the disk below the third body has not been reported. Rupture of this disk would presumably

produce pain in the side of the neck and in the clavicle, supraspinatus region, and shoulder tip.

Second Cervical Disk—The syndrome of rupture of the disk below the second body has not been determined but it is possible that some cases of chronic occipital neuritis are caused by a ruptured second disk. Young[18] mentions a case of rupture of the disk below the second body which was proved by myelography and at autopsy. The patient had stiffness of the neck followed by pain in the side of the neck and elevation of the tongue, presumably because weakened infrahyoid muscles failed to fix the hyoid bone.

ROENTGEN-RAY STUDIES

Roentgenologic findings are of great assistance in the differential diagnosis of pain in the neck, shoulder, and arm, but can be misleading. Probably the most constant finding in cases of rupture of a lower cervical disk is loss of the normal curvature,[19] even when the patient is attempting to hold the neck in hyperextension (fig. 4). Roentgenographic evidence of narrowing of a disk is a useful finding provided it is the disk which the examiner believes, on clinical grounds, to be ruptured. However, numerous verified cases have been reported in which the roentgenograms failed to demonstrate loss of disk substance. Narrowing of several disks is a frequent roentgenographic finding and many persons who are entirely symptom-free have roentgenographic evidence of one or more narrowed cervical disks and the associated bony spurs. Oblique views of the cervical spine are useful in demonstrating narrowing of intervertebral foramina and encroachment on the foramina by proliferative bony changes, and should be made in all cases of chronic pain in the neck, shoulder, and arm.

I have had no experience in demonstrating rupture of cervical disks by means of myelography with iodized oil but some utilize this method.[17,20] Murphey,[21] who has been opposed to myelography for the localization of ruptured lumbar disks, has found that myelography at the cervical level is extremely accurate.

INCIDENCE OF DISK HERNIAS

Bradford and Spurling[9] state that cervical disk ruptures constitute from 4 to 8 per cent of all disk ruptures. This estimate is presumably based on operative cases. My own figures indicate that less than 1 per cent of the disks coming to operation, excluding those that compress the spinal cord, are in the cervical region. Perhaps the true figure will eventually be between 4 and 8 per cent when

ruptured disk is routinely considered in the differential diagnosis of pain in the neck, shoulder, and arm. It might not be inappropriate to suggest at this point that ruptured thoracic disk will in time be

Fig. 4: (Case 3.) Preoperative film showing loss of normal cervical curvature.

considered in the differential diagnosis of chronic thoracic and abdominal pain, including the so-called intercostal neuritis.

SURGICAL TREATMENT

Although large protrusions of the cervical disks which damage or threaten to damage the spinal cord require fairly extensive hemilaminectomy or bilateral laminectomy, the small lesions which produce the monoradicular syndrome are best approached through a small opening immediately over the compressed nerve root. Removal of the bone usually necessitates use of a small chisel and Kerrison punch (fig. 5). If the surgeon is forced to explore two

or more roots in search of the cause of the radicular pain, he may make separate openings over each root or may enlarge the original
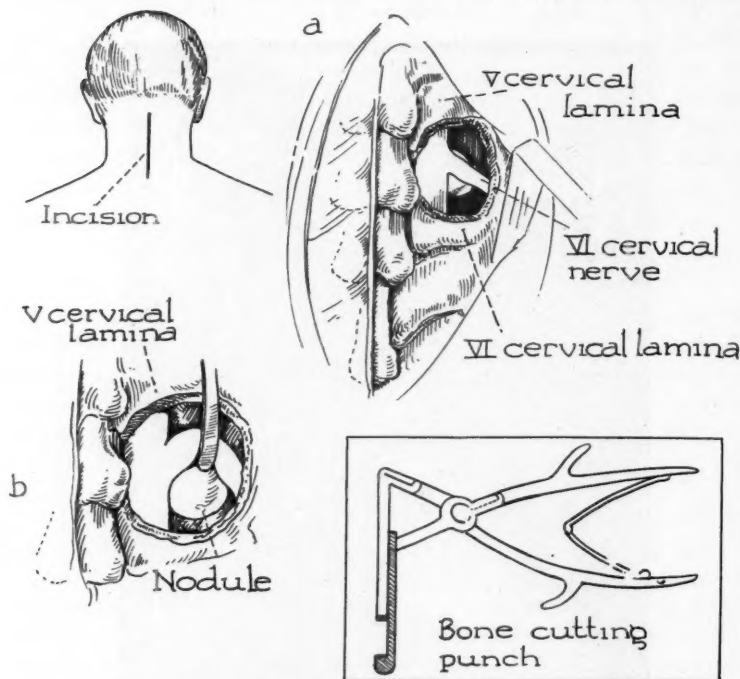

Fig. 5: Partial hemilaminectomy gives adequate exposure of the laterally placed nodule and the compressed nerve root.

opening (hemilaminectomy) until both or all three roots in question have been exposed.

DISCUSSION

Personal communications from Semmes and Murphey[22] in 1940 stimulated my interest in the possibility of operating on cervical disks which were producing the radicular syndrome. At first, I believed that these lesions would prove to be common and that the operation would be performed frequently. Now after 5 years I find that I have seen only 18 such cases and have operated on only 3 of them. The others were treated by orthopedic surgeons who were able to alleviate the symptoms by such measures as traction and immobilization. The 3 patients operated on were returned to me after conservative orthopedic measures had failed. Similar ex-

perience regarding the infrequent necessity of surgical intervention has been reported by Elliott and Kremer.[17] By means of myelography and other methods they verified 12 cases of compression of the radices by ruptured cervical disks. None of the patients were operated on and all are said to have become reasonably comfortable with conservative therapy. Young[18] reports 56 cases, only 5 of which were treated surgically.

SUMMARY

1. The several types of ruptured cervical disk have been classified according to the neural structures which they compress and 3 examples of spinal cord compression with bilateral root compression or stretching have been described.

2. The signs, symptoms and surgical treatment of disk ruptures which merely compress a nerve root have been reviewed and 3 case reports have been recorded. Similar cases in the literature have been cited.

3. It is suggested that lateral ruptures of cervical disks are fairly common but that only an occasional case requires surgical treatment.

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THE SURGICAL TREATMENT OF ACUTE CHOLECYSTITIS

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Introduction

WHILE acute cholecystitis is rather generally recognized as a surgical disease there is still a real controversy as to the proper time for operation. The trend is towards earlier surgery but the division of authorities is such that one may follow either school of thought and have ample precedent to sustain him. Because of this difference of opinion individual surgeons are more likely to be guided by their personal experiences and to express their opinions more freely than is generally the practice.

This paper is a plea for more prompt surgical intervention in the usual case.

In regard to the type of surgery indicated, cholecystectomy, rather than cholecystostomy, is the generally accepted procedure unless there are definite contraindications. The reasons for this preference will appear later in this discussion.

Pathology

The selection of the proper treatment as regards both procedure and time carried out, is based largely upon an appreciation of the pathologic changes involved.

If one includes the obstructions due to inflammatory edema, practically every case of acute cholecystitis begins as an obstructive lesion. If, by definition, one limits the term to those obstructions where there is an impacted stone present in the cystic duct, even then the vast majority of cases will fall into this category. In either instance the essential thing to remember is that primarily infection does not play an important part in the pathologic process. This is the reverse of what occurs in acute appendicitis.

With the development of obstruction an increased intravesical pressure appears. The contents of the gallbladder at that time will depend somewhat upon the length of time since the cystic duct has been patent. Whether there is fairly normal bile, white bile, or mucus is entirely incidental.

Due to the increased intravesical pressure there will be interference with the blood supply of the wall of the gallbladder. Up to and including part of this stage regression may occur. Generally the course of the disease to this stage requires from 2 to 3 days and a dissecting edema about the gallbladder quickly develops. Later this primary edema is followed by induration of the gastro-hepatic tissues, with resulting poor visualization of the involved structures.

Once the impairment of the blood supply of the gallbladder wall becomes advanced, gangrene of some degree is likely to occur. Even then in many cases the wall may remain intact or be supported by surrounding adhesions.

In others perforation will follow and by then infection will usually be present. It should be borne in mind, too, that all perforations will not necessarily involve the peritoneal cavity. Some may occur through the gallbladder bed and extend into the liver substance with the development of disastrous multiple abscesses.

If the peritoneal cavity is involved the process is usually localized by adhesions of omentum or adjacent viscera but there is always the possibility of its becoming generalized. While it is admitted that bacterial peritonitis may not be common, a chemical peritonitis due to the presence of free bile may be quite serious as will be well appreciated by any who have seen instances where the ligature on the stump of the cystic duct has slipped in cholecystectomy cases which were not drained.

Such are the progressive pathologic changes generally admitted to occur in the course of an acute cholecystitis. Differences of opinion as to treatment are based upon individual judgment as to the rapidity of the changes, the likelihood of the development of the more dangerous stages, the technical difficulties presented at operation, the appraisal of the patient's general condition, and other factors of lesser importance.

The appearance of jaundice may be the result of a toxic or infectious hepatitis, edema or stone of the common duct, or, later, multiple abscesses of the liver. Fortunately all of them are uncommon —which accounts for the absence of clinical jaundice in the usual case. Common duct involvement is said to occur in less than 20 per cent of the cases, which is a fact of some importance in determining the type of operative procedure.

CLINICAL COURSE

The attack may in its beginning resemble an ordinary colic. However, the pain is apt to be more localized, the nausea and vomiting more severe, and the tenderness and spasm in the right upper quad-

rant more pronounced. The development of fever of 101° or over, and the elevation of the white count to 12,000 or more will be of great aid in differentiating the two conditions. The development of a tender palpable mass is rather pathognomonic. Unfortunately this latter generally means that nature has lost its fight to relieve obstruction by dislodgment of the impacted stone or to decrease the acute inflammatory process. Other evidences of an extension of the disease include an increasing pulse rate, spreading rigidity in the right upper quadrant, further elevation of the white count and general deterioration of the patient.

If the clinical-course of the disease could be appraised with accuracy much of our controversy regarding treatment would be eliminated. Unfortunately, such is not the case. The symptoms, abdominal signs, and laboratory findings do not always indicate the severity of the process. Decrease of pain may mean the development of gangrene as well as a regression of the acute changes. One is often incorrect when he attempts to anticipate the course of an individual case of appendicitis. Still more confusion exists in regard to patients with acute cholecystitis. Even cases of supposedly chronic cholecystitis with cholelithiasis will occasionally show acute changes at operation.

In spite of this uncertainty, a considerable majority of cases treated by watchful waiting will subside. However, even if this does result, one can hardly call the treatment effective. Certainly the patient has not been cured. The cause of the disease is still present, future attacks more severe in character are rather certain to follow, economically there is a waste of time and money as the disability in non-operative treatment may extend over a considerable number of days, and, a point often not considered, the patient may acquire an unwarranted confidence that will result in a false sense of security and therefore may delay in securing adequate treatment in subsequent attacks.

OPERATIVE TREATMENT

Here, as in most diseases, the first measure in treatment is the prevention of the condition. Our limited knowledge of the etiologic factors, as well as our recognition of the difficulty of controlling those we know, makes it discouraging to attempt to eliminate or even greatly to reduce the incidence of gallbladder disease. Even after it is established all must admit that non-surgical treatment may be quite effective in many cases. However, when the presence of cholelithiasis is confirmed it is my conviction that the case is no longer medical. Not only are the vast majority of cases of acute

cholecystitis initiated by the presence of a stone in the cystic duct, but also nearly every other serious complication of gallbladder disease follows stone formation. Such complications include pancreatitis, choledocholithiasis, malignancy, etc. The early removal of gallbladders containing the so-called silent or innocent stones, as well as those causing rather typical colics, will not only definitely lower the incidence of acute cholecystitis and the complications just mentioned but will also thereby indirectly lessen the mortality of biliary tract disease in general.

In discussing the various times for surgery the terms "immediate," "early," and "delayed" are the ones usually employed. All of these terms are susceptible to various definitions and limitations. If by "immediate" one means within just a few hours, as an acute appendix is usually cared for, or with only a minimum of preparation or observation, most surgeons will agree that such urgency is unwise. Most debate revolves around "early," which is usually taken to mean some time in the first 3 days of the disease (not in the first 3 days of hospitalization), with provision for adequate preoperative care, and "late," which means any time subsequent to that, and also includes the interval between attacks.

One's decision should be influenced by a consideration of the following facts which have been rather well substantiated by those who have presented them:

Those cases which subside satisfactorily without early operation are the ones which would have only a minimum surgical mortality if operated upon at the time.

Those cases which do not subside will show an increasing mortality proportionate to the delay in reaching surgery.

The fulminating cases and those in the aged will have the highest surgical mortality but they will also have the highest non-surgical mortality as they are the least likely to be benefited by continued medical treatment.

Gangrene, perforation, and peritonitis rarely occur before the fourth day.

Delay generally adds to the technical difficulties of operation and makes for a higher percentage of cholecystostomies. This is undesirable as these cases generally require a subsequent cholecystectomy and with that additional operation there will be inevitable additional mortality.

When operation is deferred subsequent attacks may occur unexpectedly in the waiting interval.

With each succeeding attack there is statistically an increasing mortality.

The mortality of perforated ulcers and acute appendicitis was reduced only by early operation—should not the same apply to acute cholecystitis?

A short period of observation can be well utilized as the non-operative treatment is essentially applicable for preoperative preparation. During this time 5 per cent glucose in saline may be administered intravenously and vitamin K may be given hypodermically. My preference is for Synkamin in 1 mg. doses three times a day. Theoretically the latter may not be indicated if the clotting time and prothrombin values are normal. Tests for the latter are not always readily available and are more expensive than the drug itself. For these reasons its routine use appears to be justifiable. The temperature and pulse should be determined and recorded every 4 hours. Frequent abdominal examinations should be made and the patient's general condition carefully evaluated. The efficacy of cold applications is questionable but their use can do no harm. Narcotics should be used sparingly to avoid masking of possible warning pain. I have not used penicillin during this stage but intend to do so. Although infection is usually not prominent at that time it may become an important factor later, and my observation of the use of this drug in the early treatment of war wounds prior to the onset of suppuration has convinced me of its value. However, because of their possible deleterious side-effects I reserve the sulfonamides until I feel that infection is truly present.

If, during this period of observation and treatment, signs point to a decrease of the inflammation and particularly if jaundice or other indications point to possible common duct involvement which will require exploration of that duct, one may feel justified in deferring operation.

Generally speaking, though, I believe these cases, as well as the ones showing progression as indicated by increasing fever and pulse rate, the appearance of a tender mass, or a general failing of the patient, should be operated upon after this preliminary period of preparation. Much more will not be gained by further treatment and much may be lost from the standpoint of morbidity and mortality by further delay. Not the least of the difficulties arising will be the increased technical difficulties of the operation.

The type of anesthesia selected for any operative procedure on the gallbladder is likely to be determined by the individual surgeon's preference. All kinds of local and general anesthesia have their

proponents. My preference, unless definitely contraindicated, is for cyclopropane alone or with a few drahms of ether. The supplementary use of crurare has been enthusiastically recommended but I have found it unnecessary and have had no experience with this agent.

For years I persisted in using some type of longitudinal straight or curved incision in gallbladder surgery. After struggling with difficult closures and having some incisional hernias I was persuaded to adopt the oblique subcostal incision. It has proved most satisfactory for visualization of the biliary tract, and also the pylorus. Further, the appendix, too, can be delivered, usually without much difficulty. Closure is much easier, postoperative discomfort is considerably lessened, deep breathing is better tolerated and the resulting scar is apparently much stronger.

Because of frequent leakage of bile from the gallbladder bed and the rare but disastrous leakage from the ligated stump of the cystic duct, I prefer to use a cigarette drain. It does not appear to affect morbidity or comfort adversely and is certainly an effective exit for any biliary drainage that may occur.

In some instances cholecystostomy may have to be done. Particularly in late cases the edema may be largely supplanted by an induration that makes identification of involved structures extremely difficult, or there may be palpable stones in the common duct inaccessible because of the patient's poor general condition or technical difficulties.

Ordinarily, and particularly in the early cases, there is an edema present which provides a water dissection that facilitates the removal of the gallbladder. Many gallbladders which can not be satisfactorily or safely excised from below upwards will often be suitable for removal from above downwards. Preliminary aspiration may be necessary in the occasional case. Unless palpable stones are present in the common duct, exploration of that tract is not recommended.

Whenever possible cholecystectomy is preferable to cholecystostomy. Gallbladders non-functioning before or during the acute attack are not likely to resume function after cholecystostomy. They remain a constant threat and, even if removed before another attack develops, present the double mortality of two operations. Another argument in favor of primary cholecystectomy is the smoother convalescence shown by most of the cases so treated.

In any case the procedure of prime importance in either chole-

cystectomy or cholecystostomy is the removal of the impacted stone
when one is present.

Postoperatively, as well as preoperatively, intravenous glucose
in saline is probably the most important measure in treatment.
Vitamin K should be resumed where indicated. Penicillin and sulfa-
diazine will not be necessary unless infection is present in the peri-
toneal cavity or develops later in the lungs. When either jaundice
or sepsis appears transfusions may be necessary. Deep breathing
should of course be encouraged. Frequent change of position, in-
cluding the use of the semi-sitting posture, add to the patient's
comfort and safety. The routine use of prostigmin will help to
control postoperative distention.

CONCLUSIONS

While it is realized that every case must be individualized and
that inflexible rules can not be established for the routine treatment
of all cases of acute cholecystitis, there are certain general conclu-
sions that seem quite logical.

Acute cholecystitis is for a variable unpredictable time a progres-
sive disease.

The pathologic progress of the disease does not always conform
to the clinical signs and symptoms.

In the vast majority of cases calculi are present. Even when calculi
are absent, the pathologic changes are such that recurrence is to be
expected.

The morbidity and mortality of surgery in acute gallbladder dis-
ease are directly proportional to the delay in operating after the
onset of symptoms.

The mortality also increases with each succeeding attack before
surgery is accomplished.

During the first few days of an attack the water dissection result-
ing from the edema facilitates rather than hinders the technic of
cholecystectomy.

In that same time gangrene, perforation, and peritonitis are rare.

Proper preoperative treatment requires only a relatively few
hours.

Exposure is quite adequate when an oblique subcostal or trans-
verse incision is used.

Operation will be necessary sooner or later to secure an ultimate
cure, as non-surgical "cures" are practically never permanent.

The postoperative course after cholecystectomy is frequently smoother than that after cholecystostomy.

Since cholecystectomy offers the only permanent cure it is the procedure of choice except where it is technically unsafe or where suspected pathology in the common duct cannot be adequately determined or corrected.

Therefore, given a patient with an attack of acute cholecystitis, it is recommended that during the first 24 hours he is seen he be given intravenous glucose, vitamin K, and other supportive preoperative measures. Unless all signs point to a progressive improvement, cholecystectomy with drainage through an oblique subcostal incision on the second or third day is the most effective and efficient treatment for the average case. Even those cases showing early improvement may well be treated in a similar manner in order to secure a permanent as well as a prompt cure.

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MECKEL'S DIVERTICULUM
Report of Five Cases

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N the course of didactic training, one is familiarized with this condition from the beginning of his medical career. This knowledge is frequently displaced by active participation in routine practice. Fortunately it is possible to recover from some dusty crevice of the mind recognizable information which must be refreshed. For that reason, it seems this subject is worthy of consideration.

In 1812 Meckel first discussed the clinical significance of this diverticulum when he gave the first accurate description and embryologic background. However, according to Curd,[6] the earliest case of this diverticulum was mentioned by Lavater in 1672. Since, many reports have been made and the average incidence of various authors is about 2 per cent at postmortem table.

Meckel's diverticulum is a malformation of the gastrointestinal tract due to the persistence of an embryonic anlage—the omphalo-mesenteric duct. This duct connects the vitelline sac in the fetal membranes of the embryo with its abdominal cavity. The persistency of this duct may manifest itself in many ways. It is located on the lower ileum from 0.5 to 2.0 meters above the ileocecal valve. The length of the diverticulum varies considerably. Usually it is only a few centimeters in length and may be only a sacciform bulge. On the other hand, diverticuli up to 50 centimeters long have been described. It originates on the ileum opposite the mesentery. However, exceptions to this may occur. Wellington[2] in his report of 326 cases found 3:1 in the male.

The original entodermal continuity of the intestinal mucous membrane into the omphalomesenteric duct may be supplemented by mesenchymatour parts of the intestine. Such replacement is usually seen in diverticuli which hang freely from the intestine but this condition may reach the umbilical cord. In such cases, the formation of enterocystomas may result. Also the previous uninterrupted connection between the omphalomesenteric duct and the intestine may become strangulated or the same interruption accomplished by ligation of the umbilical cord after birth. After the remainder of the cord becomes separated, there may persist a permanent connec-

tion from the diverticulum to the umbilicus, thus resulting in a fistula. Further, if the duct becomes obliterated at its origin from the intestine, a patency of the remainder may persist. This interruption of patency may be incomplete and the persisting part of the duct become cystic. These cysts filled with mucoid material may become quite large and their origin identified only by microscopic study.

Various classifications of Meckel's diverticulum may be found. The one given by Dubs[10] is most accepted.

1. Umbilical duct diverticulum—the omphalomesenteric duct within the abdominal cavity as far as the umbilical ring has been obliterated but remains patent within the umbilical cord, resulting in umbilical granuloma.

2. True Meckel's diverticulum—the omphalomesenteric duct has undergone involution with the exception of a stump averaging about 9 centimeters which maintains a connection with the ileum.

3. Enterocystoma—the duct is obliterated toward the umbilicus and the intestines but remains open and cystic between.

4. Fistula—due to patency of the omphalomesenteric duct in its entire length.

Numerous modifications of these forms occur and are discussed by various authors.

The presence of transplanted tissue in Meckel's diverticulum is sufficiently frequent to justify theories about their origin. Since three of the cases in this report showed the presence of gastric mucosa, theories as to its origin in ectopic foci will be discussed briefly: (1) That it is due to transplantation of tissue from the original site during embryonic life, (2) that it is due to metaplasia of tissues during either embryonic or post-natal life and (3) that it is due to an atavistic phenomenon.

There have been many schools of thought concerning the types of transplantation of these tissues. Misplaced gastric mucosa may be frequently found in the esophagus and Meckel's diverticulum. On the other hand, misplaced intestinal mucosa is never seen within the esophagus or stomach. The orally situated parts of the gastrointestinal tract dominate in this respect the distally located ones. Curd[6] suggested that gastric mucosa became engrafted and subsequently grew in the vitelline duct. Branch and Gross[7] postulated the possibilities of inclusion of primitive cells in the foregut. Mitchell and Angrist[5] expressed the theory of inclusion of epithelial cells from the foregut. The transposition theory seems most acceptable.

Metaplasia according to Boyd is the transformation of one type of tissue into another type, but this process has definite limits in-

sofar as only those tissues arising from the same germ layer can replace each other. As regards metaplasia Taylor[8] regarded heterotopic tissues as dysontogenetic structures, that is, structures arising in the individual organism from abnormal differentiation of the embryonic entoderm under abnormal stimulation. Kind and McCallum[9] disagreed with this theory and stated that ectopic tissue developed during postnatal and even adult life and felt that inflammation may lead to metaplasia of tissues. They further showed that ectopic tissue is mixed with the tissue of the host and frequently is found at the site of inflammation. This theory of metaplasia is not accepted by most authors.

According to Lauche[14] the atavistic heterotopias are mainly found within the lymphatic apparatus of the intestines. This reverts to a more primitive phylogenetic type such as seen in certain lower animals and fishes and hardly seems plausible as explanatory of heterotopias pathologically.

Clinically, Meckel's diverticulum most often remains quiescent and is found most frequently as a non-significant post-mortem finding. The true diverticulum may assume all stages of inflammation. In its mildest form it produces only slight mid-abdominal cramps, with or without nausea. In this stage, it cannot be entirely differentiated from acute appendicitis. Because of abnormally located tenderness it may be suspected. In case of operation, if a normal appendix is found after suspicious history of acute appendicitis, the distal 2.0 meters of the ileum should be carefully explored. It should be suspected as cause of melena in children. Diagnosis after roentgenologic demonstration has been reported by Tragey and Adams.[1] They took hourly roentgenograms until the head of barium meal reached the cecum. Because of its fixation to the anterior or posterior abdominal wall, it may be the cause of acute intestinal obstruction, due to herniation of bowel through a fixed loop or volvulus about a fixed point. In either case the diverticulum is usually inflamed and requires removal. It is frequently involved in, and the exciting factor of, intussusception.

In the extreme form it may become gangrenous and result in acute peritonitis. Where there has been persistent ulceration, especially in children, acute perforation may occur as a very dramatic affair, usually accompanied by heterotopic tissues, either gastric or pancreatic. Two such cases are reported in this paper.

Foreign bodies may lodge in the diverticulum producing either acute inflammatory changes or perforation. The symptoms will vary according to the pathologic process. Various tumors have been

reported as occurring, such as sarcoma, carcinoma, hemangioma and myoma.

It can be seen that the symptoms may be very different, depending upon the type of anomaly, the degree of inflammation, or presence of heterotopic tissue. In cases operated upon with suspicious history of acute appendicitis in which a normal appendix is found, diligent search should be made for Meckel's diverticulitis. Also when cause for general peritonitis is unknown, it should be remembered that Meckel's diverticulum may be the offending member.

The following cases have come under my observation:

CASE 1: C. B. C., Jr., aged 3 years, white male, was seen in consultation with Dr. Charles Kennon on Aug. 25, 1943, at 3:00 P.M., complaining of generalized abdominal pain. About noon the child desired evacuation and, while at stool, screamed, complaining of acute abdominal pain. This was followed by nausea and associated with pallor. He was seen by Dr. Kennon, who found evidence of shock and a very tender abdomen. Immediate blood count revealed white cells 10,500 with 80 per cent neutrophils. Urinalysis was negative.

Upon my arrival the child was lying on his right side with his legs drawn up and crying because of abdominal pain. The temperature per rectum was 98 deg. F. and pulse was 110 per minute, regular but thready. It was quite evident some dramatic abdominal accident had occurred. The abdomen was rigid and very tender upon palpation with most marked tenderness around the umbilicus. No mass could be felt upon abdominal or rectal examination. There was no evidence of peristalsis and no blood evidenced per rectum.

The child entered the Jackson Memorial Hospital at 8:30 P.M. Immediate x-ray study of flat and upright abdomen revealed small amount of free gas beneath both diaphragmatic leaves, more marked on the left. There were numerous small fluid levels present throughout the mid-abdomen. Some gas was present in colon and also in proximal small bowel. Roentgenologic diagnosis was perforated hollow viscus, site not apparent.

The patient was a strong, healthy child. Temperature was 99.8 deg. F. per rectum and pulse 115. There was rigidity of the abdominal muscles and evidence of tenderness throughout the abdomen but especially around the umbilicus. The remainder of the physical examination gave negative results. The urine was reported as negative. Hemoglobin was 70 per cent, red blood cells numbered 3,600,000; white blood cells, 21,450; with 87 per cent neutrophils, 8 per cent lymphocytes and 5 per cent monocytes.

A diagnosis of intussusception or ruptured Meckel's diverticulum was made. Plans were made for immediate surgery. Under vinyl ether anesthesia and novocaine block a right paramedian incision opposite the umbilicus was made at 10:00 P.M. Upon entering the peritoneal cavity a large amount of cloudy fluid exuded. The intestines were reddened and distended. A leaking sound could be heard upon manipulation and was found to arise from a perforated Meckel's diverticulum located on lower ileum about 1 metre proximal to the cecum. It was adherent to the mesentery from which it was freed. Its wide base required longitudinal excision from the ileum. The defect was closed transversely, using two rows of #0 chromic catgut sutures, overlaid by inter-

rupted #C silk sutures. Sulfathiazol (micro crystals) 5 grams was spread in abdominal cavity around operative site.

The postoperative course was uneventful, the wound healed primarily and subsequent health of child has been good.

Pathologic examination: Gross: Specimen consists of a pouch covered with serosa. It is cone-like in appearance, measuring about 4 centimeters in height and about 2 centimeters at its base. On one side there is a perforation measuring about 0.5 centimeters in diameter. The wall is up to 1 centimeter thick.

Microscopic: Section shows the mucous membrane to consist of typical epithelium usually seen in the ileum but the area of the perforation and the edges of the perforation show typical prepyloric gastric mucosa. The edge of the perforation is formed by a characteristic ulcer usually seen in the stomach, the surface of which consists of fibrin, pus cells, lymphocytes, plasma cells, etc. From this ulcer on both sides, and towards the serosa, there is a large amount of inflammatory infiltration noted and the evidence of numerous eosinophilic leucocytes is somewhat striking.

Diagnosis: Meckel's diverticulum with heterotopic mucous membrane, the latter ulcerated and perforated.

CASE 2: P. P., age 14 years, white male, was seen by Dr. George Williams about 10:00 A.M., Jan. 10, 1945, complaining of abdominal cramps, with pain radiating into groins and testicles on both sides. This had begun during the preceding night and had become progressively worse. He had vomited once during the night but there was no persistent nausea. There was no history of previous gastrointestinal upsets.

Physical examination: There was tenderness with spasm over lower abdomen, more marked in right lower quadrant, and bilateral rebound tenderness. Rectal examination was negative except for tenderness. Other findings were essentially negative. The temperature was 99.6 deg. F., blood pressure 120/70. Urinalysis was negative. White blood cells were 15,000 with 92 per cent leucocytes.

Patient was admitted to Edgewater Hospital with a diagnosis of acute appendicitis. Examination upon entering hospital revealed more tenderness over left than right lower abdomen.

Operation: The abdomen was entered about 1:00 P.M., through a McBurney muscle-splitting incision. An apparently normal appendix was removed without difficulty. The terminal ileum was brought up into the wound, over it and on its mesentery were found flakes of yellowish exudate. About one metre from the ileocecal valve a Meckel's diverticulum was found covered with exudate and appearing gangrenous. It was about 2.5 centimeters across at its base and 2 centimeters long. Near its base was a perforation. It was dissected free from the mesentery to which it was adherent. A straight clamp was placed across its base and then amputated. It was closed with 2 layers of continuous #0 chromic catgut sutures. Sulfathiazol powder 5 grams was spread around this area within the peritoneal cavity. The abdominal wall was closed without drainage.

Postoperative course was uneventful and patient discharged on 10th day. General health since has been good.

Pathologic examination: Gross: Specimen consists of an irregularly shaped piece of intestinal tissue which is split and the previous structure is not determinable any more. Only microscopic sections will determine whether one is dealing with mucous membrane of the intestines or whether there is an inflammatory tumor mass present. Appendix routinely removed.

Microscopic: Section of the ruptured diverticulum shows the border lines to be built up by typical mucous membrane belonging to the small intestines. The edges are undermined by a typical mucous membrane belonging to the structure of the stomach with all the characteristics of these cells. The area of ruptures is in this misplaced gastric tissue.

Diagnosis: Ruptured Meckel's diverticulum with misplaced mucous membrane of the stomach.

CASE 3: A. N., white male, about 63 years old, entered Jackson Memorial Hospital Sept. 19, 1942, complaining of abdominal cramps, tightness in lower abdomen, and constipation. He was under the medical care of Dr. A. J. Logie.

Several years previously he had an appendectomy with subsequent drainage and extensive scarring in right lower paramedian region. Since that time he complained of the above symptoms, with increase in severity, associated with nausea and vomiting occasionally. He had had marked aggravation of symptoms in past three weeks.

Physical examination revealed a well nourished white male in no acute pain. Blood pressure was 130/90, pulse 88. Heart and lungs were otherwise negative. In the right lower abdomen there was tenderness, especially upon pressure, over the right paramedian scar. There was increase in peristalsis and some high-pitched sounds. X-ray examination showed few loops of small intestine in right lower abdomen. Barium enema revealed spastic colon. Urinalysis showed trace of albumen and few pus cells. Hemoglobin was 90 per cent; red blood cells 4,440,000; white blood cells 12,750 with 76 per cent neutrophils. Provisional diagnosis was partial intestinal obstruction. High enema resulted in passage of much flatus and fecal material. Regardless of these results, it was decided to prepare for elective surgery. This was done Sept. 23, 1942.

Operation: Under cyclopropane anaesthesia and novocain block, the old right paramedian scar was removed. With difficulty the abdominal cavity was entered. Loops of ileum were adherent with many contortions and partial occlusion. In the scar also a protrusion from the ileum was buried. After careful dissection about 10 centimeters of an elongated Meckel's diverticulum was delivered. Its base was narrow and it was clamped, ligated, excised, and stump inverted without occlusion of lumen. Abdomen closed without drainage.

Postoperative condition was good and the course very satisfactory until the fifth day when he suddenly went into circulatory failure. Flatus had passed, the abdomen was soft and no signs of peritoneal involvement seen. The internist felt the diagnosis of acute myocardial failure was justified. He died within 12 hours in deepening collapse in spite of all conceivable supportive measures.

Pathologic findings microscopically showed marked exfoliation of the superficial layers of epithelum. The interglandular tissue was densely infiltrated with lymphocytes and a few pus cells. Though a few glands presented a typical regeneration, there was nowhere evidence of malignancy.

Diagnosis: Inflamed diverticulum.

CASE 4: P. I., age 18, a white male, entered Hall Hospital June 15, 1937, under the care of Dr. K. Phillips complaining of acute abdominal pain. He had taken ill 12 hours previously with severe abdominal cramps associated with excessive vomiting. In spite of intravenous glucose he had become worse. I saw him in consultation and advised immediate surgery.

Physical examination was negative except for the abdominal findings. The abdomen was distended and typmonitic. There was hyperperistalsis with high pitched tinkling sounds. Extreme tenderness was present in the lower abdomen and generalized spasticity of the abdominal muscles. Rectal examination revealed no masses. Blood findings were normal except for leucocytosis. Diagnosis of acute intestinal obstruction with vascular changes was made.

Operation was performed under ether anesthesia. A right paramedian incision opposite the umbilicus was made. Upon entering the peritoneal cavity distended loops of ileum were evident. There was a herniation of a loop of ileum through an opening between a fixed loop of ileum and the posterior abdominal wall. This point of fixation was found to be a gangrenous Meckel's diverticulum. Upon its mobilization the continuity of the intestinal tract was reestablished. The diverticulum was about 11 centimeters long and had a narrow base. The base was clamped and ligated. The diverticulum was then excised and the stump inverted. The abdomen was closed without drainage.

There was moderate peritoneal inflammation which subsided under conservative care. The patient healed primarily and left the hospital on the 14th day. Subsequent health has been good.

CASE 5: E. C. C., white male, aged 68 years, entered Jackson Memorial Hospital Feb. 1, 1946, as attempted suicide with stab wound of chest. His history is not related to this report, the patient having generalized arteriosclerosis and coronary changes, and dying a cardiovascular-renal death on March 2, 1946.

In the course of routine postmorten examination, a Meckel's diverticulum was found with two pouches.

Preliminary microscopic study shows misplaced gastric mucous membrane in smaller diverticulum.

SUMMARY

A brief review of history and origin of Meckel's diverticulum has been given. Theories relating to heterotopia of gastric and pancreatic tissue has been discussed, with the opinion that transplantation seems the most plausible explanation.

Three cases of Meckel's diverticulum have been presented, showing heterotopia of gastric mucosa. Two revealed ulceration with perforation.

One case had been operated previously for acute appendicitis. Because of the extensive scarring, with adhesions and partial obstruction, and because of the presence of the inflamed diverticulum in the scar, it is quite likely the inflammation of Meckel's diverticulum was present at the first operation and had persisted in sub-

acute form ever since. The cause of death in this case is still un-explained. For five days the postoperative course was uneventful. Permission for postmortem examination could not be obtained.

The fourth case presents the possibility of obstruction from Meckel's diverticulum. In such a condition there are usually in-flammatory changes in the diverticulum itself.

The fifth case was found incidentally at routine postmortem ex-amination, revealing double diverticuli from common origin with heterotopic gastric mucosa.

Three phases of clinical manifestation are illustrated by cases. All five cases occurred in males.

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PERSONAL EXPERIENCES IN THE SURGICAL TREATMENT OF GASTRIC AND DUODENAL ULCER

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THE essentials of this discussion are based largely upon our experience with the surgical treatment of gastric and duodenal ulcer. The tendency toward resection has been progressive throughout the years, until now subtotal gastrectomy is the operation of choice when the conditions are favorable for this procedure. Definite indications have been established for operation in both gastric and duodenal ulcer, based upon certain complications and malignant potentialities.

GASTRIC ULCER

Chronic gastric ulcer is distinctly a surgical lesion more because of penetration, hemorrhage and obstruction, than because of the occasional association of malignant degeneration. Local excision, with or without gastroenterostomy, is inadequate and a poor substitute for subtotal gastrectomy. The latter procedure is attended with a minimum mortality, has none of the difficulties often encountered in duodenal ulcer, and gastro-jejunal ulcer rarely develops subsequently.

We have had no mortality in resection for gastric ulcer and uniformly results are more satisfactory than resection for duodenal ulcer. There have been no stomal ulcers.

Lesions high on the lesser curvature or at the gastroesophageal junction cannot be resected except by total gastrectomy, an undesirable procedure. Excision often is anatomically impossible and an indirect attack does not adequately reduce gastric acidity.

Colp and Druckerman[1] report favorable results by palliative subtotal gastrectomy distal to the lesion, thereby sufficiently reducing gastric acidity with resulting healing of the ulcer. A procedure of merit perhaps, except, perchance, the lesion is malignant, which cannot always be determined even by inspection and palpation.

Gastric ulceration of recent development must at all times be considered an ulcerative carcinoma until proven to be benign. There is no positive criterion by which the true nature of an ulcerative lesion of the stomach may be determined. As mentioned by Walters[2] and Sanders,[3] many early ulcerative carcinomas of the stomach

give benign symptomatology and respond for a time adequately to medical management. The location of the lesion gives some information, but it is inconclusive. Lesions in the immediate prepyloric region, on the greater curvature and in the fundus, are to be considered malignant. Those on the anterior and posterior wall have a malignant potentiality of 20 per cent; and along the lesser curvature, 10 per cent. Gastric acidity is of no great value in differential diagnosis as it is found in both benign and early malignant lesions. Perhaps excessive acidity indicates simple ulcer. Gastric acidity, in reality, is only an index to the activity of the exciting cause, the probable acuteness of the ulcerative process, a guide to prognosis, and an index to the type and extent of treatment.

Time is a factor, and a period of only 3 to 4 weeks should be permitted for study. If, at the expiration of that time, the lesion has not healed, as demonstrated by competent radiologic study, by gastroscopic observation, and by the fact that blood has not disappeared from the stomach contents and the stool, the lesion should be considered either intractable to medical management or an early ulcerative carcinoma. The lesion should be excised and its true nature determined. If benign, an intractable lesion has been removed before complications of fixation and penetration have occurred, and, if malignant, a better opportunity for cure has been provided.

DUODENAL ULCER

Ulcer of the duodenum is largely a self-limited disease, subject to remissions and exacerbations. Sometimes a permanent remission occurs, as evidenced by case histories and autopsy findings. Usually, however, the disease is of long standing, accompanied by remissions and relapses, frequently resulting in perforation, obstruction, hemorrhage or intractable indigestion.

PERFORATED DUODENAL ULCER

We have not attempted, at the time of operation for perforation, any procedure other than the simplest that was necessary to save the patient's life. These patients are in no physiologic or anatomic condition for any procedure directed at the cure of the ulcer. Their lives are in danger and efforts should be directed to get them through alive. Any further surgery, such as gastroenterostomy, or subtotal gastric resection, adds materially to the risk. Only when there is a distinct constriction of the pylorus after closure should a gastroenterostomy be done—never, in our opinion, such an extended procedure as subtotal resection.

After closure and recovery, many will not need further surgery but will be sufficiently comfortable under medical management. At any rate, a resection can be done at a later date under more favorable conditions, when the problem of life and death is not such a factor.

That perforations do recur is often observed and, after the second perforation, resection should be done in a reasonable period of time. In five years we have operated upon 6 patients by subtotal gastric resection who had had two or more perforations of a duodenal ulcer, one with four. One had had two perforations within two weeks of a gastrojejunal ulcer. In this type of perforation I am sure that simple closure is often unsafe because of inaccessibility, excessive local induration and fixation. The gastroenterostomy should be taken down, the stomach and jejunum closed, and subtotal resection deferred to a later date, if the pylorus is patent.

PYLORIC OBSTRUCTION, COMPLETE AND PARTIAL

Complete pyloric obstruction from scar tissue represents the end results of a burnt-out peptic ulcer. The patient is starved, emaciated, suffering from hypoproteinemia and vitamin deficiency and in a poor physiologic and anatomic state for major surgery. I think it is unwise to make a definite statement that all these patients should have a subtotal gastric resection. Each case must be considered individually and no unnecessary risk should be assumed. If the patient is old, has a short life expectancy and low acid values, I prefer to assume the risk of a subsequent gastrojejunal ulcer and do a gastroenterostomy, rather than the more extended procedure of resection. This is the one indication for gastroenterostomy, and I know of no procedure quite so satisfactory.

On the other hand, if the patient has a relatively long life expectancy, high gastric acidity, and greater physiologic reserve, he should have a subtotal gastric resection.

There is another group of lesions associated with recurrent edema that obstructs from time to time but responds adequately to hospital medical management, only to obstruct again under the requirement of their daily life and improper dietary conditions. After three such episodes these patients should have a resection, as it is only by this procedure that they will be able to resume their routine existence.

BLEEDING FROM DUODENAL ULCER

I speak of bleeding from duodenal ulcer because most ulcers are duodenal. The situation is unchanged, regardless of whether the

ulcer is gastric or duodenal. Distinction must be made between massive and repeated recurring hemorrhage. If massive, the decision for or against operation must be made early, within 36 to 48 hours. Operation after that period has a mortality which increases as the hours pass and it soon becomes prohibitive. Sudden massive bleeding is so exsanguinating that the patient is in poor condition for major surgery, even though repeated transfusions are given. That patients do die from hemorrhage there can be no doubt.

Within the last year we saw one patient die, at the age of 42 years, before surgery could be instituted. However, one at 65 years of age was given multiple transfusions and operated upon on the second day, and survived a subtotal resection. Two others with less severe hemorrhages survived early subtotal resection. This is a problem that requires judgment and regardless of the decision all too often one wishes he had done otherwise.

The operation in the face of hemorrhage is difficult. The stomach is full of blood, the tissues are stained and access to the bleeding point is often difficult because of the low position of the ulcer. A mattress suture of silk around the bleeding vessel in the ulcer bed is sometimes all that can be done. Resection of the ulcer is desirable but unwise if induration and ulceration extend low in the duodenum near the common bile duct.

Hemorrhage in a young individual is not a surgical problem but with increasing years it becomes more serious and after 50 years of age surgery should be done for chronic recurring bleeding. A patient with a history of hemorrhages over several years, or frequent hemorrhages over a period of a year or two, in our judgment, should be operated upon before a massive hemorrhage occurs.

Intractable Ulcer or Intractable Chronic Indigestion

I believe that intractable indigestion is an indication for operation, even in the absence of bleeding or obstruction. One must be sure that the lesion is intractable because of a large crater, indicating penetration into the head of the pancreas. The psychogenic factors must be evaluated and, if prominent, operation should be deferred. Bloomfield[4] states that if they seem inconsequential, the operation of excision of a scarred, deep ulcer, with reconstruction by a wide stoma, is sound practice, even if bleeding or obstruction are absent. I have seen many patients who had such intractable indigestion from a deep calloused ulcer that any degree of comfort was almost impossible. Our experience with excision of this type of ulcer and high resection has resulted in a gastric comfort not experienced for many years.

CLINICAL RECORD

In this series under survey we have performed 91 subtotal gastric resections for ulcerative lesions of the stomach, duodenum and the gastrojejunal stoma. In the group there were 13 gastric ulcers, 6 with two or more previous operations for perforated duodenal ulcer, 7 for gastrojejunal ulcer, 3 for massive extensive hemorrhage, and the remainder for major complications of duodenal ulcer.

There were 3 deaths in this series following operation, one from peritonitis from rupture of the duodenal stump, one from a cerebral accident, and one from coronary thrombosis. The duodenal rupture was occasioned by insecure closure following resection of a large indurated duodenal ulcer, in which an exclusion type of operation would have been preferable. The cerebral lesion developed in a hypertensive elderly individual upon whom a gastroenterostomy would have been more desirable, and the coronary thrombosis probably could not have been prevented by less radical surgery.

There were 5 major complications associated with this group which required early recognition and prompt treatment. One, with bronchial occlusion, relieved by bronchial aspiration; one had a subhepatic abscess which was surgically drained; two developed small bowel obstruction, released by operation; and the fourth, with obstruction of the proximal limb of the anastomosis resulting in an enormous distention of the duodenum, recovered after anastomosis was made between the duodenum and the jejunum.

In addition, there were 31 posterior gastroenterostomies for benign obstruction in elderly individuals, most of which were done early in this series. There was one death, due to non-functioning stoma, found to be non-obstructive on secondary exploration.

There were 11 perforations of duodenal ulcer with one death— the patient was not operated upon until 36 hours after perforation.

There were, in addition to this group, 12 cases of carcinoma of the stomach upon whom we did a subtotal gastrectomy, the early clinical history being that of a benign gastric ulcer. Each had received symptomatic relief for a time. Only 5 had had a radiographic study confirming an early ulcerative lesion. All were diagnosed gastric carcinoma when first seen by us. Clinically it is to be assumed that they were primary ulcerative carcinomas, giving the symptomatology and therapeutic response of a benign ulcer, but whose pathology at the time of operation was so advanced it could not be proved. There were no operative deaths.

Of the ulcer group we know of but one stomal ulcer; many of

the patients could not be located. The results from subtotal gastric resection have been about comparable to those usually obtained by medical treatment in the uncomplicated ulcer. A 10 per. cent group remains incompletely relieved, with a variable degree of gastric distress.

SURGICAL PROCEDURES FOR ULCERS

Surgery for gastric and duodenal ulcer may be divided into three types: the conservative; the resection; and the vagotomy.

The conservative is represented by pyloroplasty and gastroenterostomy; the resection, by pylorectomy, antrectomy and subtotal resection; and the vagotomy by section of the vagus nerves.

We never were impressed by the procedure of pyloroplasty, and we do not do it. Gastroenterostomy has been a controversial subject. It remains to this date, however, the most frequent stomach operation and the safest. We have abandoned this operation except in elderly individuals with pyloric obstruction. We believe the procedure unsuitable for non-obstructive ulcer—the incidence of stomal ulcer is too frequent.

Because of this serious objection, efforts were made by aggressive surgeons to eliminate this complication by more radical surgery. The European surgeons, principally Von Harberer and Finsterer, pioneered the field, followed by Berg, Lewissohn and Strauss in this country. Finsterer reported in 1926 that in the preceding 7 years he had performed 593 partial gastrectomies for ulcer of the stomach and duodenum, and gastroenterostomy only 5 times.

RESECTION

The first attempts at resection were limited to the pylorus and antrum, with inadequate results and with similar complications of stomal ulcer. Failure stimulated a bolder approach with more extensive resection until now the accepted procedure is to extend it to the point of near acid reduction. Thus, with an adequate resection, removal of the pylorus, a good stoma and an uninterrupted inlet and outlet, the incidence of stomal ulcer is small. Wagensteen[5] emphasizes the necessity of a short proximal loop and Lahey[6] insists that the length of this loop is not important and that it should be long and in the ante-colic position. Lannin[7] speaks of tissue resistance to acid secretion and says that it is reduced with the length of the proximal loop. Also, that secretin, a hormone, is diminished in the jejunum as distance lengthens from its origin, and without secretin the gastric contents do not stimulate the secretion of alka-

line pancreatic contents and thus stomal ulcer is more likely to occur. His report of 241 cases shows no stomal ulcer with a very short proximal loop type of anastomosis, while Kiefer,[8] from the Lahey Clinic, reports an incidence of 6.9 per cent in 173 cases. However, an ante-colic anastomosis has many advantages. It is more accessible for accurate suture, the anastomosed limbs are less likely to become constricted by angulation from the mesocolon, and gastrojejunal ulcer, when it does occur, is more approachable for secondary operation. Gastrojejunal-colic fistula does not occur in the ante-colic procedure. We have, for many years, employed the ante-colic anastomosis of the Hofmeister end-to-side type with good results. We do not do an enteroentero anastomosis.

Resection should include the ulcer when anatomically possible. The location of the ulcer in relation to the common bile duct should be determined and the amount of duodenum available for secure closure ascertained. This is best done by exposing the common bile duct and determining its position in relation to the ulcer. With exposure the duct will not be injured. This is easily done and it is a satisfaction to know its position. With the duodenum open, an inserted finger can guide the dissection of the indurated ulcer from its crater bed.

Exclusion Operations

The Finsterer exclusion operation is a procedure preferable to gastroenterostomy if the ulcer cannot be removed because of induration and fixation. It is essential, however, to remove the antral mucous membrane completely to abolish acid secretion from contacting the ulcer bed. We have employed this in 11 cases successfully, without evidence of subsequent ulceration.

Gastrojejunal Ulcer

Gastrojejunal ulcers may be treated by medical management and a few will respond; often, however, with subsequent contraction and stenosis of the stomal opening. Our experience, like that of Sanders,[3] has been that they are intractable to medical measures and require disconnection and resection. We believe that gastric resection should always accompany the disconnection of a gastroenterostomy for jejunal ulcer.

Those associated with a previous gastric resection will frequently heal by pylorectomy if the previous operation had been one of exclusion. If not, further resection should be done. Associated with a colic fistula, the procedure is more complicated and attended by

excessive mortality. Our experience is inadequate to express a preferable opinion, but reference is made to Lahey's[6] right colectomy two-stage operation and to Ranson's[9] procedure.

VAGOTOMY

Vagotomy is a recent and as yet a relatively unevaluated procedure reported by Dragstedt[10] and others. It is performed transthoracically for the purpose of reducing gastric acidity with resulting ulcer healing. Results are reported favorable in selected cases. We have had no experience with the procedure. For the complications of ulcer to which I refer and for which I advocate direct or indirect surgery, it seems to be a most indirect and inadequate approach. It holds promise, by its acid reduction factor, of being a distinct aid in the medical management of uncomplicated ulcer. Since we have no personal experience, we shall not be the first to adopt the new nor the last to discard the old.

SUMMARY

1. Opinions concerning indications for operation of gastric and duodenal ulcer have been expressed.

2. Early operations in gastric ulcer suspected of possible malignancy is emphasized and urged.

3. Surgical experience, results and procedure employed in a relatively small group of cases are recorded.

4. Discussion of the surgical procedures most desirable for the individual case is presented and the advantages and disadvantages are emphasized.

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PARTIAL HEPATECTOMY IN CERTAIN PRIMARY TUMORS OF THE LIVER

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RIMARY tumors of the liver are regarded by most surgeons with an air of futility and in most instances, when found at the time of operation, the incision is closed with no more being done than a biopsy. Because of the scarcity of recent literature upon this subject and as the result of a personal experience with a large primary tumor of the liver, I am presenting this paper as an effort to point out the favorable aspects for removal of such lesions in order that these individuals will not be denied their only chance for cure. Since hepatic tumors are not usually diagnosed preoperatively, the surgeon is frequently unprepared to undertake a resection if he has not previously had experience with such a case. In many of the resections which have been reported the end results would have been much better had the operator been familiar with the various methods available to accomplish this somewhat hazardous procedure.

The literature on this subject has recently been reviewed by Warvi and he presents a classification of clinically significant primary tumors of the liver based upon histologic study. As seen (fig. 1), these fall under four main headings: hepatoma, cholangioma, cholangiohepatoma, and tumors primary in the liver but not containing specific hepatic elements. Liver cell adenomas are usually single, well encapsulated, and consist of cords of atypical liver cells with intervening blood sinusoids. There are no bile ducts or portal triads and no evidence of blood vessel invasion. A true liver cell adenoma is physiologically and anatomically completely isolated from the normal liver tissue. The gross cut section of the tumor is of a pale greenish color due to the fact that there are no bile ducts present to carry away the bile produced by the liver cells. No attempt will be made to delve into the controversial subject of hamartoma except to say that these are small, slowly growing rests of liver cells which resemble adenomas pathologically and clinically. It is very important to differentiate between true liver cell adenomas and the nodular hyperplasia of cirrhosis. The latter are not surrounded by true capsules and are connected with the duct system of the liver. The liver cell carcinoma, or malignant hepatoma, may

occur either with or without cirrhosis. In many instances carcinoma arises from a liver cell adenoma. It may appear as a single massive growth or as multiple nodules with or without cirrhosis. The liver cells are small with a loss of cytoplasm and rapid cell proliferation. The signs of secretion, such as gross bile staining, which are present in adenoma, are entirely absent in liver cell carcinoma. There is little doubt that cirrhosis plays a considerable role in the etiology of carcinoma, as it was found to be present in 46 per cent of the cases of primary liver cell carcinoma at the Cincinnati General Hospital. Other authors report its association in 85 to 100 per cent of the cases.

Cholangiomas arise in the biliary ducts and may appear as a benign adenoma or a carcinoma. The adenomas are in most instances cystadenomas, but in rare instances appear as the solid or tubular type. A malignant cholangioma appears as adenocarcinoma, cystadenocarcinoma, or as carcinoma simplex, in which case it is usually associated with cirrhosis of the liver. Cholangioma is more common in females, and this may be due to the increased frequency of bile duct infection. Cholangiohepatoma consists of both liver cell and duct elements. The duct structures grow more rapidly, and therefore are predominant over the liver parenchyma cells. The fourth group consists of tumors which do not contain specific hepatic elements. These may consist of tumors of vascular origin; hemangioma, lymphangioma, and endothelioma; adrenal rest tumors; sarcoma; or teratoid tumor. Of these, hemangioma is by far the most common, and frequently is represented as small tumors, but in many instances reaches quite large size, and rupture of a hemangioma of the liver into the peritoneal cavity is not uncommon.

The resectability and prognosis depend upon the type of tumor, its location, its size, and the general condition of the patient. Pedunculated growths and those situated on the anterior edge of the liver are accessible and usually relatively easily resected. Growths near the hilum or on the under surface of the liver or high under the diaphragm offer considerably more difficulty and in some instances are unresectable. If the tumor is benign, such as the liver cell adenoma, the benign cholangioma, hemangioma, or fibroma, the prognosis is excellent if the tumor has been completely resected. Warvi found 33 cases of resection of solid adenoma of the liver in the literature and postoperative mortality was 9 per cent. There was recurrence of the tumor in 21 per cent within one to six and one-half years postoperatively. Seventy per cent were living and well from two and one-half to five years after removal of the

hepatoma. He reported three additional cases resected at the Cincinnati General Hospital. One of these patients, in whom the adenoma could be only partially removed, died of recurrence three and one-half years later, and the other two were living and well eight and one-half years and two years respectively after operation.

Even in malignant primary growths of the liver the prognosis is much more favorable than would be expected. Metastasis from

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      PRIMARY TUMORS OF THE LIVER

      1.  Hepatomas
          a.  Liver cell adenomas
          b.  Liver cell carcinomas
      2.  Cholangiomas
          a.  Adenomas of intrahepatic bile
              ducts, solid or cystic.
          b.  Carcinomas
      3.  Cholangiohepatomas-both liver and
          cell duct elements.
      4.  Tumors primary in the liver but
          not of specific hepatic elements
          (Vascular, fibrous, adrenal rests,
          etc.)
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Fig. 1. Classification of primary tumors of the liver.

carcinoma of the liver is usually limited to within the liver itself and extra-hepatic metastasis is not frequent. When it does occur it is usually found in the lungs, mediastinum, and upper abdominal lymph glands. Yeoman in 1915 collected from the literature 16 cases of primary carcinoma of the liver which had been resected. Four of these died in the postoperative period, six died from recurrence, ranging from two months to eight years after operation, and six were alive and well from three to seven years after operation. In other words, 37.5 per cent of these 16 patients had no evidence of recurrence at intervals varying from three to seven years after operation. This is particularly significant when we consider that the disease is rapidly fatal when no treatment is given. In 1939, 14 additional cases of malignant tumor of the liver were reported by Charache. The follow-up period upon this group of patients is not as long, but the figures are encouraging. Two of the patients died postoperatively and two died within two years of recurrence.

One of the patients was not followed after discharge. Eight of the 13 patients upon whom follow-up data was available (61.5 per cent) were alive and well from seven months to nine years after operation. These statistics would indicate that in solitary primary carcinomas of the liver resection is not only feasible but definitely indicated if the patient's condition will warrant. It is generally felt that resection is justified in adenomas, cystadenomas, localized solitary carcinomas, and hemangiomas, provided there is no lymph node metastasis at the hilum and the growth does not involve the major blood vessels or ducts.

My interest in this subject was inspired by a recent case upon whom I performed a complete lobectomy for a large hepatoma of the left lobe of the liver. An abstract of this case is presented:

A 14 year old white girl, A. J. K., was examined at the Wheeling Clinic on April 21, 1945. She stated that she had noticed a hard lump in the upper left quadrant of the abdomen for about one year. Her only other complaint was that she tired easily and had noticed a dragging sensation in the abdomen. Upon physical examination the significant findings were limited to the abdomen. In the left upper quadrant was found a mass approximately four inches in diameter, the upper border of which could not be definitely made out, as it appeared to extend under the costal margin. It was somewhat movable and progressed downward upon deep inspiration. The border of the right lobe of the liver was just palpable beneath the right costal margin.

She was admitted to the Wheeling Hospital on June 21, where various laboratory tests were performed. Hemoglobin was 82 per cent, red blood cells 4,160,000, and white blood cells, 8,200. Differential count showed lymphocytes 32, neutrophils 64, basophils 4. Serologic test for syphilis was negative; platelet count, 282,880; coagulation time, 4 minutes; bleeding time and fragility test, normal; total plasma protein, 6.9 Gm. per cent.

It was felt preoperatively that this tumor was an enlarged spleen and represented either a cyst of the spleen or an early Banti's disease. Operation was advised and carried out on June 25, employing cyclopropane anesthesia. A left transverse incision was made below the left costal margin and this was later extended just beyond the midline, and then upward as a right paramedian to the xiphoid process. A round, irregular tumor was found occupying almost the entire left lobe of the liver. It was adherent only to the omentum. The right lobe of the liver appeared entirely normal. The spleen lay above and to the left of the tumor and was likewise normal. There was no enlargement of the hylar lymph nodes. It was felt that resection was indicated, and lobectomy was carried out as follows: The left triangular ligament was cut close to its attachment to the diaphragm, and the falciform ligament was similarly severed. This mobilized the entire left lobe of the liver and it could be drawn into the incision. The left hepatic artery, the left branch of the portal vein, and the left hepatic duct were ligated with suture ligatures of chromic catgut. A row of mattress sutures of chromic catgut was then placed along the interlobar sulcus in such a manner that each suture overlapped the adjacent one. These were placed by passing a long, straight needle in a vertical direction through the liver substance along the relatively

avascular line of union of the two lobes. The left lobe of the liver was then removed by cutting with a scalpel along the line just to the left of this row of sutures. Following this procedure there was no bleeding from the cut surface of the liver and there was extremely little loss of blood during the

Fig. 2. Cut section of left lobe of liver containing large heptoma.

entire operative procedure. There was also no spillage of bile. A gauze pack was placed against the cut liver surface and brought out the upper end of the incision. The abdominal incision was then closed in layers, using interrupted sutures of silk on the fascia.

Figure 2 shows the cut section of the left lobe. The specimen measured 13x9x9 cm. and was almost completely occupied by the tumor. It weighed 535 Gm. The tumor itself was 10 cm. in diameter and the tissue was solid and tough on section. The cut section of the tumor was stained a light green, due to retained bile secreted by the liver cells of the tumor which had no access to the surrounding liver. The microscopic appearance of the tumor is shown (fig. 3) where the liver cells are found to be arranged in cords and round islands resembling lobules. The tumor cells are separated by bands of dense pink connective tissue and bile ducts are not formed. Under high power the large, flat liver cells produce a finely granular, pink cytoplasm and some contain bile pigment. The nuclei vary in size, and some are hyperchromatic. Mytotic figures are not found. Pathologic diagnosis was hepatoma or primary benign liver cell adenoma.

The patient's postoperative course was essentially uneventful. She was given one blood transfusion during the operation and postoperatively 10 per cent glucose intravenously for several days. The gauze packing was removed gradually, beginning on the first postoperative day, and was completely removed by the fifth. There was no drainage of bile from the incision and no bleeding. Her highest temperature reading was 102 degrees on the first post-

operative day, and became normal by the fifth day. A small amount of infection developed at the upper end of the incision where the pack had been placed, but this soon cleared, and she was discharged from the hospital twenty
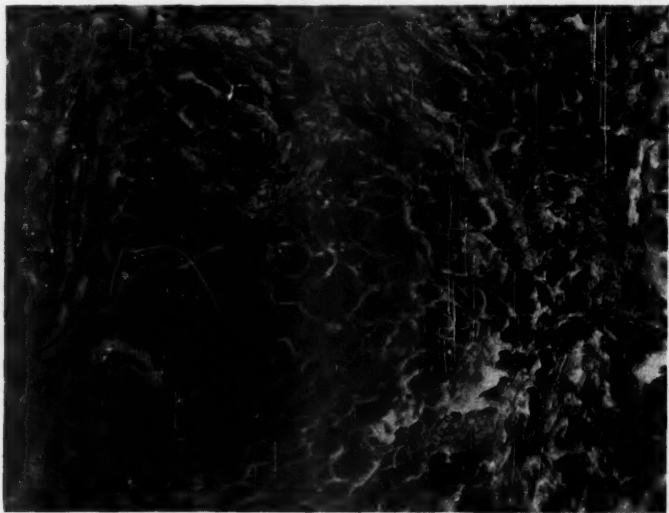

Fig. 3. Microscopic section of heptoma. Note the absence of ducts and hepatic triads.

days after the operation. When the fall term of school opened six weeks later she resumed her school work, and at the present time is in fine health and has gained ten pounds in weight.

A recent review of world literature revealed only 33 reported cases of resection of solid adenoma of the liver, but there were 223 cases of resections of liver tumors. Since any surgeon may at some time be called upon to resect a portion of the liver, let us consider the various methods by which this has been carried out. Tumors situated in the left lobe of the liver are in most cases best approached by performing a lobectomy, except for the very small tumors. By carrying the line of resection along the interlobar sulcus, a comparatively avascular area, the problem of hemostasis is greatly reduced. It is felt that the best method to accomplish this is that which has just been described, employing mattress sutures through the liver substance previous to resection of the lobe. Pickrell and Clay, in 1944, found only two reports in the literature where there has been a total removal of the left lobe of the liver, and they reported three other cases of their own at the Johns Hopkins Hospital. It was formerly thought that removal of so much of the liver would result in decreased liver function. However, the powers of

regeneration of the liver are very great. Turner, in 1923, removed an adenoma along with a considerable amount of hepatic tissue weighing two pounds and three ounces, and the patient suffered no impairment of liver function. The greatest problem in resection of portions of the liver is that of hemostasis. Usually the most applicable method is that of the placement of mattress sutures along the contemplated line of incision before the incision is made. Some have advocated cutting the liver tissue with the cautery as a further method of reducing bleeding. Cousins, in 1874, recommended placing an elastic ligature around the stump, and then extraperitonealizing the tumor with subsequent removal. Others have used clamps across the liver edge with removal of these clamps several days later. In 1897 Von Rosenthal controlled bleeding in resecting a pedunculated angioma of the liver by placing knitting needles across the superior and inferior surfaces of the liver and compressing the liver tissues between them by placing ligatures through the tissue around the needles. The latter are brought out either through the incision or through a stab wound and are removed several days later. Some of these tumors can be extraperitonealized and the stump sutured to the peritoneal edges of the incision. Several writers have excised portions of the liver using only the cautery, but it would seem that the danger of postoperative hemorrhage is too great to recommend this procedure. When a wedge-shaped portion of the organ is removed it is frequently possible to approximate the two cut surfaces with mattress sutures after the bleeding has been controlled. Any sudden hemorrhage from the liver may be controlled temporarily by compression of the hepatic artery and portal vein in the duodenohepatic ligament.

SUMMARY AND CONCLUSIONS

Primary tumors of the liver should be resected if the lesion is localized and favorably situated and if there is no evidence of metastasis to regional nodes or other organs. Tumors situated in the left lobe of the liver are more easily and safely removed by lobectomy. A case report is given wherein a complete removal of the left lobe of the liver was performed upon a 14-year-old girl for a large hepatoma, and the various methods for accomplishing hemostasis are presented. The liver has a great capacity for regeneration and therefore large portions of it may be removed without deleterious effects upon liver function. Partial hepatectomy can be performed safely and with a good prognosis in many instances.

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THE MANAGEMENT OF THE RUPTURED
APPENDIX

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Richmond, Virginia

THE mortality from appendicitis is still too high. Out of every 100,000 of population, over 10 people will die from appendicitis this year. When we realize that there is no mortality from appendectomy while the organ is unruptured, one becomes very disturbed about this high mortality rate.

It has been definitely shown, and generally adopted, that the appendix should be removed before the inflammation has extended beyond the appendix itself. A prompt appendectomy should be done, therefore, for acute appendicitis. There is very little agreement, however, on the proper treatment to follow if the appendix ruptures.

When an appendix ruptures, one of three stages of peritonitis develops:

1. Localized peritonitis

2. Localized abscess

3. Spreading or diffuse peritonitis

The duration of the disease, the length of time which elapses before the patient comes to the hospital, the resistance of the patient, and the virulence of the offending organisms, all play a part in determining which stage will be present in a given patient.

The chief cause of death in peritonitis is the intestinal obstruction which follows the paralytic ileus. Theoretically, any method of treatment which would prohibit the development of ileus should materially reduce the mortality from peritonitis.

In 1940 we radically changed the manner of treating cases of appendiceal peritonitis, according to the suggestion of Finney, of Baltimore. He advocated the postoperative use of castor oil and aspiration of the infected material from the peritoneal cavity. The results obtained by this treatment were so dramatic that its use has become routine in our hospitals. The castor oil is, of course, used in addition to the other main supportive measures used prior to

1940 and has become a necessary adjunct to the successful treat-
ment of peritonitis.

I am sure that all of you in this distinguished gathering have
seen patients die with generalized peritonitis from a ruptured ap-
pendix, in spite of any treatment you might give, because of a stead-
ily increasing abdominal distention. The introduction of castor oil,
by stimulating peristalsis, prevents this increasing ileus. As soon as
a bowel movement occurs, these patients make a rapid recovery.

In our preliminary report published in 1942,[1] we showed how the
mortality of diffuse appendiceal peritonitis had been reduced from
12 per cent to 1.3 per cent by the addition of castor oil to our form
of treatment.

TABLE 1

SUMMARY OF 3,000 CASES OF APPENDICITIS AT THE MEDICAL COLLEGE OF VIRGINIA 1931-1941

	cases	*died*	*mortality*
Acute	2028	11	0.54%
Abscess	428	27	6.3
Diffuse peritonitis	471	57	12.1
Peritonitis—castor oil used	73	1	1.3

We had used castor oil in 73 cases at that time with only one death.
Since that report we have had another 64 cases to add; and in this
latter group, we had only one death. The two deaths in 137 cases
gives us a mortality rate of 1.4 per cent.

This presentation is limited to the cases we encountered at the
Medical College Hospital between July, 1941, and July, 1945.
There were 186 cases of perforated appendix of all types. There
were 68 cases of localized abscess, 75 cases of localized peritonitis,
and 43 cases of generalized peritonitis. In the entire group there
were 7 deaths for an overall mortality of 3.7 per cent.

Castor oil was originally given only to those patients having a
generalized peritonitis, but it has now been used to some extent in
the other forms of peritonitis. This group of 186 cases revealed
that of the 64 patients treated with castor oil, 32 had generalized
peritonitis, 20 had local peritonitis and 12 had appendiceal ab-
scesses. In the 43 cases of generalized peritonitis there were four
deaths, a mortality of 9.7 per cent. Eleven of these patients did
not receive castor oil. Three of these patients died, a 27.2 per cent

mortality. Of the 32 patients who did receive castor oil, only one died. This latter group has a mortality of 3.3 per cent. There were no deaths in cases of localized peritonitis or abscesses treated with castor oil.

TABLE 2

THE EFFECT OF THE CASTOR OIL TREATMENT ON CASES OF DIFFUSE PERITONITIS

	cases	*died*	*mortality*
Diffuse peritonitis	43	4	9.7%
Castor oil used	32	1	3.3
Castor oil not used	11	3	27.2

APPENDICEAL ABSCESSES

In this series there were 68 abscess cases, 19 of which presented a palpable mass on admission (28 per cent). We believe in treating palpable appendiceal abscesses with "watchful waiting." This conservative treatment is followed as long as the patient improves —as indicated by a decrease in the size of the abscess, a reduction in temperature and leukocyte count. On the other hand, if there is no improvement, we recommend prompt drainage. The appendix is not removed at the time of drainage unless it is lying free in the abscess cavity. An extraperitoneal drainage is done wherever possible, the pus is evacuated by suction and cigarette drains inserted. The wound is not sutured.

It is obvious from our figures that the greater part of appendiceal abscesses are not palpable on admission. Even though an abscess is suspected, because of the duration of symptoms and the physical findings we do a prompt operation in these cases. If the abscess is early, we remove the appendix and treat it as a case of localized peritonitis. If the abscess is well formed, the free peritoneal cavity is packed off, the abscess carefully opened, the pus evacuated, and the cavity drained.

Thirty-four of our 68 abscess cases were operated upon on admission. One of this group died. Twenty-five of the patients had to have simple drainage, and of these, 2 died. Of the 9 cases treated conservatively, none died. Conservatism, here, is the best policy.

All 3 of the deaths in cases of appendiceal abscess occurred in 1941, early in our series. Since that time we have not had a death in 59 consecutive cases.

If appendix is not removed at the time of drainage, we have the patient return in 6 weeks for an interval appendectomy. Some patients, particularly the colored ones, will not return, either because they feel too well and see no reason for being operated upon again, or they conveniently "forget" to return. Fortunately, this

TABLE 3

MORTALITY RATE IN APPENDICEAL ABSCESS ACCORDING TO MANNER OF TREATMENT

	cases	died	mortality
Appendectomy alone	34	1	2.9%
Drainage with or without appendectomy	25	2	8.0
Conservative	9	0	0.0

group is small. Of the 23 in this series who did not have their appendix removed, either because of conservative treatment or because of inaccessability at the time of drainage, only 7 never returned to our hospital.

LOCALIZED PERITONITIS

In all other types of appendicitis, that is, in all cases in which we do not feel a mass, we do a prompt appendectomy. This group includes all cases in which there is a localized area of peritonitis, but not yet a walling off to form an abscess. There were 75 cases in this group. None was fatal. Twenty patients were ill enough to require the castor oil treatment, but it is not as often necessary in this group as in cases of generalized peritonitis. We use castor oil in the localized cases where there has been a considerable amount of handling of the bowel to deliver the appendix, or when, because of contamination of a larger area of the peritoneal cavity, we feel that ileus may present a serious problem.

GENERALIZED PERITONITIS

In the 43 cases of generalized peritonitis in this series there were four deaths. While the resulting mortality of 9.7 per cent compares favorably with other published series, it is still too high.

All cases in this group are submitted to prompt operation. The appendix is removed, and, wherever possible, the stump inverted. We then carefully aspirate as much of the pus and debris as it is

possible to remove. From 7.7 to 10 grams of a mixed sulfanilamide and sulfathiazole powder is inserted into the peritoneal cavity, and the wound is closed without drainage. We condemn to oblivion the use of drains in generalized peritonitis. Even though many surgeons are still following the age-old dictum of "when in doubt, drain," it has been clearly shown by Cottis[2] and others that the mortality from peritonitis when drains are used is three times as great as in those cases in which drainage is not used. Elman and Eckert,[3] among others, have abandoned the use of drains and we agree with Ochsner and Johnston[4] that drains should not be used unless there is a localized abscess, danger of hemorrhage, or an unremoved, localized necrotic area.

Drainage of the abdominal wall, however, must be done. We treat the abdominal wound in one of two ways. If there is no gross spilling of pus into the layers of the wound, we close it around drains carried down to the peritoneum. If there has been gross soilage of the edges of the wounds, we thoroughly irrigate them with saline solution, insert retention sutures through the skin and muscle fascia, place several grams of the mixed sulfonamide powder into the wound, and pack it open. After three or four days, when the danger of infection is over, the pack is removed and the wound edges approximated by tying the sutures. We have never had any bad effects from such a plan of wound treatment and we have practically eliminated the incidence of wound infections.

All patients suspected of having peritonitis are subjected to the insertion of a nasal tube into the stomach before operation. As soon as the patient is back in his room, suction drainage is instituted and the stomach emptied. We then insert an ounce of castor oil through the tube and clamp it off for two hours. The tube is then reopened and the patient allowed to drink water as soon as he desires. He is given parenteral fluids and vitamins, whole blood transfusions, and an ampoule of prostigmine every three to four hours. Following the work of Fine,[5] we give many of these patients high concentrations of oxygen. At the end of eight hours, the dose of castor oil is repeated and the tube again clamped off for two hours. At the end of another eight hours, if the bowels have not moved, we give an ounce of milk of magnesia through the tube every hour for two or three doses. If there is still no bowel action, we give a small six ounce enema to instigate the first passage. Once the bowels have moved, we note a marked improvement in the patient. His abdomen softens, his temperature subsides, he feels much better, and he is no longer acutely ill.

Prior to the adoption of castor oil to treat peritonitis of appen-

diceal origin, our mortality in generalized peritonitis was 14.8 per cent in over 400 cases. Since the use of castor oil it has dropped to 1.4 per cent. It must be stated that some of the cases in this series were treated with castor oil prior to the introduction of the sulfonamides, and almost all of them before the adoption of penicillin.

TABLE 4

COMPARISON OF CASES TREATED WITH AND WITHOUT CASTOR OIL OVER A 10 YEAR PERIOD 1934-1945

	cases	died	mortality
Without castor oil	359	53	14.8%
With castor oil	137	2	1.4

We do not mean to imply that castor oil, alone, is the main factor in this marked reduction, but we do feel that it is a most valuable adjunct to the other necessary measures, such as whole blood, gastric suction, oxygen and the antibiotics.

There are definite contraindications to the use of castor oil: it must never be used unless the appendix has been removed and the stump inverted nor when there is a gangrenous or necrotic cecum.

TABLE 5

CONTRAINDICATIONS: CASTOR OIL TREATMENT
Never use if—
1. Appendix not removed.
2. Unsatisfactory inversion of the stump.
3. Gangrenous or necrotic cecum.
4. Associated intestinal resection.

In the past two and a half years we have not had a single death in cases having appendiceal peritonitis, in which we employed the castor oil treatment.

CHEMOTHERAPY

We were among the first to adopt the local and systemic use of the sulfonamides in the treatment of peritonitis. Our experience is that in many hundreds of cases the sulfonamides have produced no serious reactions, and we are certain that their use is clinically justified. There is little doubt in many reported series of cases that the mortality of appendicitis is less since the advent of the sulfona-

mides. We supplement the local use of these drugs with intravenous sulfadiazine after the first 24 hours, and in this series, we used sulfonamides in 86 per cent of the cases.

In July, 1944, we adopted the use of penicillin in cases of appendiceal peritonitis. Since that time we have used it in 62 per cent of our cases and this percentage is increasing all the time. We now advocate the routine use of a combination of the sulfonamides locally and parenterally and penicillin intramuscularly.

In cases of appendiceal peritonitis prior to the use of sulfonamides our mortality was 16.0 per cent. Since the adoption of sulfonamides this mortality has dropped to 5.5 per cent, although part of this drop is due to the increased use of the castor oil treatment. Since we have been using penicillin, with or without the sulfonamides, we have not had a single death in any type of ruptured appendix.

ANALYSIS OF DEATHS

Maes, Boyce, and McFetridge[6] showed some 12 years ago that appendicitis in the colored race is a more serious disease than it is in the white race. Appendicitis as it is seen in large general hospitals such as ours, and especially in those caring for large numbers of colored patients, is an entirely different disease from that seen in private practice or in private hospitals. A comparison of statistics between two such groups is futile.

Forty-three per cent of all cases of appendicitis coming into our hands are found to be ruptured on admission. This represents almost every other case. The chief cause of this percentage is the indiscriminate use of laxatives with the onset of the disease. Keyes[7] calls attention to a sensation of "gas stoppage" during the onset of acute appendicitis in 80 per cent of the cases. This sensation causes the patients to attempt to defecate or to take laxatives. Forty per cent of the patients in this series took laxatives prior to admission. This fact became more deplorable when it was found that in 25 per cent of the cases the laxative was prescribed by the local physician. Only by an increasing amount of education of the laity can we prevent the attempt to overcome this "gas stoppage" by taking laxatives, but the medical profession must take some of the blame for prescribing them in cases of abdominal pain.

We had 7 deaths in this series, all of which were in colored patients. Two died of massive atelectasis; two from a mechanical intestinal obstruction, one of which was not recognized until too late; one died because of a postoperative rupture of a gangrenous

cecum: this patient was also a severe diabetic; one died from a rupture of a large appendiceal abscess which was treated conservatively too long; and the other died because of a gangrenous cecum which prevented the use of castor oil: she developed an irreversible paralytic ileus. The only death in those patients receiving castor oil was in one of the cases of atelectasis, a feeble-minded state ward, who died within six hours after the development of the collapsed lung.

COMPLICATIONS

In this present series there has been a marked reduction in postoperative complications. Chemotherapy has greatly reduced the incidence of pulmonary complications, but we feel that the use of castor oil has been the chief factor in reducing the incidence of pelvic and subphrenic abscesses by over 50 per cent. These abscesses were twice as frequent in those patients not receiving castor oil as they were in those who received it. Apparently, the initiation of peristalsis prevents the accumulation and stagnation of infected fluid in any one area, so that the body itself, aided by sulfonamides and penicillin, can take care of the infection.

EARLY RISING

I cannot let this opportunity pass without bringing up the subject of "early rising" as was so strongly advocated by Arnold Jackson yesterday. We are adopting the regime of early ambulation more and more. We routinely get the patients with unruptured appendicitis out of bed on the first postoperative day and they are usually allowed to go home on the fourth or fifth day. I have had one patient who was out of bed in 24 hours and was notified of the death of her mother. She signed her release on the second postoperative day to attend the funeral. When she returned in a week to have her sutures removed, she admitted to very little interference in her usual activities. Although we do not advocate such radical ambulation, it does show how unnecessary prolonged bed rest is. We have adopted the "middle road" in advocating early rising.

In the ruptured cases we keep the patient in bed until he has had his first bowel movement. Then we get him up in a chair if his wound was closed at the time of operation around the drains down to the closed peritoneum. If the delayed closure was used, we keep the patient in bed until 48 hours after the sutures are tied. There is little or no reason to keep patients in bed after a simple drainage of an appendiceal abscess, most of which are drained under local anesthesia.

SUMMARY AND CONCLUSIONS

We have presented the results in treating 186 cases of ruptured appendix at the Medical College of Virginia Hospitals during the four year period ending in June, 1945. We have advocated immediate operation in all cases of appendicitis in which a definite abscess cannot be felt. We believe that appendiceal abscesses are best treated by the conservative treatment of "watchful waiting," but do a prompt drainage whenever the patient fails to improve on this regime.

We have presented further observations on the use of castor oil in the treatment of appendiceal peritonitis, by means of which, in combination with gastric suction, whole blood, oxygen, and chemotherapy, we have reduced our mortality from 12 per cent to 1.4 per cent.

Although we decry the indiscriminate use of castor oil where it is definitely contraindicated, we are convinced that it has assumed a definite place in the treatment of peritonitis. Not only is the mortality lower in cases in which castor oil is used, but convalescence is easier and quicker, the incidence of complications is reduced and the general hazards of peritonitis are eliminated.

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THE USE OF CURARE TO SECURE MUSCULAR RELAXATION IN ABDOMINAL SURGERY

Our early knowledge of curare came from its use by natives of South America in the sixteenth century, as poison-tipped arrows and blowgun darts in hunting and fighting. The victim was temporarily paralyzed, giving the hunter time either to bind the victim or to kill him at his leisure. The poison was made by brewing a concentrated mixture of native plants. Squibb & Sons now have a product on the market, "Intocostrin," which is a standard 2 per cent solution of curare. It comes in 5 and 10 c.c. rubber-stoppered bottles, ready for intravenous use.

Curare has been used in laboratory experiments for many years, but its first use in connection with anesthesia was reported by Griffith in 1942, and later by Cullen and others.

Satisfactory abdominal surgery depends upon anesthesia and proper relaxation. The term anesthesia was originally used to mean relief from pain, but modern technical usage of the word confers on it the implication of muscular relexation, that facilitates many difficult abdominal surgical problems. This relaxation was formerly obtained by a deeper anesthesia and a higher blood concentration of the anesthetic agent, which is not always safe for the patient. Some anesthetists use a combination of two or more anesthetic agents to accomplish this relaxation. For the past ten years we have used pentothal sodium intravenously with oxygen inhala-

tion as an anesthetic. It ⸱ ⸱ ⸱ ⸱nt from the patient's standpoint, efficient from the surgeo ⸱ ⸱nd safe in the hands of a properly trained anesthetist, but to secure the desired relaxation at times required a larger dose of pentothal, which kept the patient asleep for several hours after the operation was completed. For the past twelve months the anesthesia department of the Georgia Baptist Hospital has been using curare intravenously in connection with pentothal-sodium-oxygen anesthesia to secure relaxation with less anesthesia, making it safer for the patient and more satisfactory for the surgeon.

Curare accomplishes the desired relaxation by interrupting the usual nerve impulses, and its action is entirely peripheral. It affects the muscles in the following order: first, those muscles supplied by the cranial nerves, then those of the trunk and extremities, and finally those of respiration; the diaphragm is the last to be paralyzed. Subjectively, the effects on an unanesthetized and unpremedicated patient are, in order of appearance: weakness of eyelids, weakness of throat and jaw muscles, inability to swallow and to cough, inability of the patient to raise himself, weakness of arms and legs, and, finally, respiratory paralysis. Recovery is in reverse order, the diaphragm and intercostal muscles being the first to recover. The literature states that in cases where an overdose of curare has been given, resulting in respiratory paralysis, the effect lasts only a few minutes and artificial respiration can be given until the muscles of respiration recover and resume their normal function. The antidote for curare is 1 c.c. of 1:2000 solution of prostigimine given intravenously. Under anesthesia, the only effects seen, with the proper dosage, is relaxation of the abdominal muscles. Routinely our anesthetists now give intc strin, 60 to 80 mg., about the time the skin incision is made and, when the peritoneum is opened, relaxation is complete. At times we have had the intocostrin withheld until after the abdomen is opened and the intestines protrude, as they do when relaxation is insufficient, then give the intocostrin and it is spectacular in thirty seconds to one minute to see the loops of intestine literally crawl back into the abdominal cavity.

Intocostrin is a respiratory depressor but, instead of being a dangerous adjunct to anesthesia, we feel that in the hands of a properly trained anesthetist, it makes anesthesia safer and more satisfactory. Laryngopasm is less noticeable when intocostrin is used, facilitating endotracheal intubation when necessary, and it is especially valuable in bronchoscopic work.

There are two contraindications to the use of intocostrin, the first

is myasthenia gravis, and the other is the inability of the anesthetist to perform artificial respiration. The anesthetist must be absolutely certain of his ability to care for respiratory failure by performing artificial respiration, such as inflation of the patient's lungs by the use of the gas bag and oxygen, while the physiologic antidote (prostigimine) for a toxic dose of curare is administered. This antidote must always be available and ready for use.

In the past twelve months at the Georgia Baptist Hospital intocostrin has been given to more than 600 patients, mostly abdominal cases, and we have had no undesirable reaction. In a few instances, where an extra large dose has been given while closing a difficult case, one c.c. of prostigimine has been given intravenously by the anesthetist as a precaution.

Curare is broken down by the liver and eliminated by the kidneys in a very short time. Marked liver damage and poor kidney function, therefore, must be considered as a handicap in the use of curare. The initial dose of intocostrin is 60 to 80 mg. and an additional 40 to 60 mg. are given as needed to maintain proper relaxation. The effects wear off in about 15 to 20 minutes. One unaccustomed to the drug should at first proceed with caution. With experience comes the feeling of security and satisfaction. We now use intocostrin routinely in abdominal surgery, and also with special cases in general surgery. The dose of pentothal is approximately one-fourth to one-third less, the patient is not so deeply anesthetized, and awakens more promptly. We consider the addition of intocostrin to pentothal-oxygen anesthesia a definite advantage in general abdominal surgery and gynecology, and we have had no serious complications.

A more detailed paper by us on this subject will appear in this journal soon.

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NEW DIVISION FORMED TO CARRY OUT PROVISIONS OF HOSPITAL SURVEY AND CONSTRUCTION ACT

Formation of a Division of Hospital Facilities to assist Surgeon General Thomas Parran in carrying out the provisions of the Hospital Survey and Construction Act has been announced by the United States Public Health Service.

This division will absorb the functions of the Hospital Facilities Section of the States Relations Division, Bureau of State Services, which was abolished simultaneously with the creation of the Division of Hospital Facilities.

Headed by Medical Director Vane M. Hoge, the newly formed division of the Bureau of State Services will be responsible for carrying out the functions which the U. S. Public Health Service is authorized to perform in accordance with the provisions of the Act. This will include assistance to the States, their political subdivisions, and non-profit organizations in matters relating to the study, construction and operation of hospitals. The Division will also assist the Surgeon General in preparing regulations, determining allotments and grants and considering applications, plans and projects.

Allotment figures to the States for the five year hospital construction pro gram authorized in the Hospital Survey and Construction Act (Public Law 725) have been released by Surgeon General Thomas Parran, of the United States Public Health Service. The Act authorizes the appropriation of $3,000,000 for Statewide hospital surveys and for planning of construction programs, $75,000,000 annually for five years for the actual construction of hospitals and related facilities.

Of these amounts authorized, only $2,350,000 has been appropriated to date. This is earmarked for assistance to States in surveying and planning and for administrative expenses of the U. S. Public Health Service in connection with this program.

The share to which each State is entitled from the $3,000,000 authorization for survey and planning expenses is based solely on State population. For determination of the distribution of the $75,000,000 authorized for construction, a formula is used which takes into consideration both the population and the per capita income of each State.

The allotments to the several States based on appropriations authorized in the Act are contingent upon Department of Commerce certification of population data. Preliminary estimates follow:

| | ALLOTMENTS | |
State	Survey and Planning	Construction
Alabama	$ 62,422	$ 2,888,925
Alaska	10,000	40,200
Arizona	13,482	452,175
Arkansas	39,294	1,968,300
California	185,820	1,957,875
Colorado	24,279	657,300
Connecticut	40,474	421,950
Delaware	10,000	86,625
District of Columbia	19,145	298,350
Florida	47,141	1,461,900
Georgia	68,735	2,978,775

State	Survey and Planning	Construction
Hawaii	$ 10,119	$ 237,525
Idaho	10,531	293,550
Illinois	172,752	2,771,175
Indiana	77,526	1,727,775
Iowa	51,182	1,341,675
Kansas	37,908	933,750
Kentucky	57,672	2,589,600
Louisiana	53,631	2,156,850
Maine	17,671	454,875
Maryland	46,167	870,675
Massachusetts	93,515	1,595,550
Michigan	124,372	2,172,000
Minnesota	56,876	1,655,700
Mississippi	45,548	2,403,825
Missouri	79,679	2,282,550
Montana	10,355	231,825
Nebraska	26,461	685,200
Nevada	10,000	49,575
New Hampshire	10,207	342,375
New Jersey	93,928	1,313,775
New Mexico	11,210	457,500
New York	282,492	2,945,100
North Carolina	76,287	3,432,825
North Dakota	11,889	308,475
Ohio	156,144	2,692,575
Oklahoma	44,427	1,640,550
Oregon	27,317	460,875
Pennsylvania	209,243	4,551,675
Puerto Rico	46,049	2,430,525
Rhode Island	15,989	280,275
South Carolina	41,123	1,976,775
South Dakota	12,066	359,625
Tennessee	64,812	2,673,300
Texas	145,051	4,842,075
Utah	13,541	365,100
Vermont	10,000	214,725
Virginia	64,310	2,210,175
Washington	44,722	512,100
West Virginia	39,294	1,555,650
Wisconsin	67,142	1,622,925
Wyoming	10,000	144,975
	$3,000,000	$75,000,000